48

UNIVERSITY OF CALIFORNIA
APR 6 1940
LIBRARY

JOURNAL of the AMERICAN VETERINARY MEDICAL ASSOCIATION

VOLUME XCVI, NUMBER 757 APRIL 1940

TABLE OF CONTENTS, PAGE II

"CORN STATES" EVALUATES GOOD WILL

Good will built up among the guardians of animal production accounts for twenty-five years of continuous success on the "Corn States'" calendar.

★

CENTRAL LABORATORY
BUILDING PLANT
No. 1, OMAHA

★

Good will **is a precious possession and a priceless one when based upon fair dealing, equitable principles and the merchandizing of faultless products.**

Good will **enjoyed among the better element of the veterinary profession and the industry it serves establishes the pleasant business relations from laboratory to farm which provide protection to an important national resource.**

Good will **safeguarded by scientific methods of production, fair prices for all, accommodating distributors, and convenient sources of supply is the background of the "Corn States'" enterprise.**

CULTIVATING THE GOOD WILL OF THE VETERINARY PROFESSION IN THE PUBLIC INTEREST IS THE AIM OF

THE

CORN STATES SERUM COMPANY

OMAHA

Journal
of the
American Veterinary Medical Association
221 N. La Salle St., Chicago, Ill.
Copyright 1940 by American Veterinary Medical Association
VOL. XCVI APRIL 1940 NO. 757

Calcium and Phosphorus Deficiencies in Cattle and Horses: Clinical Picture, Treatment and Prevention*

By H. SCHMIDT
College Station, Texas

DURING the past 15 years much work has been done on the calcium and phosphorus requirements of lactating cows. Forbes and his coworkers[1,2] found that cows in medium milk production or even low milk production show a negative calcium and phosphorus balance which becomes positive only when the milk yield drops below 10 lbs. per day. Others report that liberally producing cows can be kept in calcium and phosphorus balance under certain conditions. In all of these experiments short-term balance experiments in the different periods of lactation were employed. With the rations containing a liberal amount of calcium and phosphorus the cow would excrete more of these minerals than it consumed, whereas when the milk yield decreased considerably or the cow went dry or nearly dry, calcium and phosphorus were stored in the body.

CALCIUM AND PHOSPHORUS IN RELATION TO LACTATION

The amount of calcium and phosphorus lost during lactation depends to a great extent upon the amount of these elements contained in the ration and upon the character of the ration itself. Thus, Miller, Yates, Jones and Brandt[3] were able to show that, other things being equal, the amount of minerals utilized from the feed was largely influenced by the character of the feed and, more especially, the roughage consumed by the cow. Fingerling[4] showed that phosphorus was more easily assimilated from green plants than from the hay made from such plants. Hart and his coworkers[5] found that the amount of calcium and phosphorus lost from the body by lactating cows was less when green grass was fed than when dried grass was given and that it was also less when alfalfa hay properly cured in the sunlight was fed than when poorly cured alfalfa hay was fed.[6]

The factors responsible for this difference in the utilization of these minerals by lactating cows are as yet not wholly understood, but it seems certain that vitamin D plays only a small part in it, for the administration of liberal amounts of the vitamin in its variously known forms, such as cod liver oil (Meigs *et al*,[7] Hart *et al*,[8]), sunlight (Hart *et al*[9]), ultraviolet-light irradiation of the cow (Hart *et al*[10]) and irradiated yeast (Hart *et al*,[11] Hess *et al*[12]), did not improve the calcium retention significantly.

Huffman, Robinson and Winter[13] have shown that cows receiving a ration of timothy hay, corn silage and grain without

*From the division of veterinary science, Texas Agricultural Experiment Station; presented at the 76th annual meeting of the A.V.M.A., Memphis, Tenn., August 28 to September 1, 1939.

mineral supplement received sufficient calcium and phosphorus for the production of at least 10,000 lbs. of milk a year. Although the cows were in a negative calcium and phosphorus balance at the height of production, positive calcium balances were obtained on this ration in cows giving 23 to 28 kg. (51 to 62 lbs.) of milk daily and receiving in the feed 42 to 56 Gm. (1.38 to 1.9 oz.) of calcium daily, with a calcium over phosphorus ratio varying from 0.88 to 0.98. When the basal ration was supplemented with 3 per cent of bone flour or calcium carbonate, positive balances were secured with most cows even during heavy production periods.

Ellenberger, Newlander and Jones[14,15] abandoned the short-period-balance experiments in lactating cows and substituted therefor balance experiments running throughout the entire period of lactation and gestation. This was a significant departure from the earlier experiments and was designed to cover the whole cycle of mineral balance of the lactating and gestating cow so that, at the end, one could determine definitely the amount of calcium and phosphorus consumed, the amount utilized in the production of milk and in the production of the calf, and the amount stored in the body. In their experiments they covered two successive periods of lactation and gestation for the individual cow. They found in cows receiving, besides concentrates, timothy hay plus freshly cut green grass in season and corn silage as a roughage, that from one calf to the next the cow stored both calcium and phosphorus. Their data showed that their cows were normally in a negative calcium balance during the first six to eight weeks of lactation, while the phosphorus balance was not always negative during the same period. When liberal amounts of mineral supplements were allowed in addition to the regular ration, the negative periods were noticeably shortened, resulting in a decided increase in the amount of calcium stored but a slight decrease in the amount of phosphorus retained. Cows yielding 26.3 to 35 kg. (58 to 77 lbs.) of milk daily could be kept in positive calcium and phosphorus balance on 95 Gm. (3.15 oz.) each of calcium and phosphorus in the feed and mineral supplement combined, and cows yielding 16 kg. (35 lbs.) of milk daily could be kept in calcium balance on 45 Gm. (1.5 oz.) of calcium daily in the feed alone.

The negative calcium balances at the beginning of the lactation period can not be attributed wholly to the amount of milk produced, as is clearly shown by the data presented by Ellenberger, Newlander and Jones, for in a number of cases the milk yield during this negative calcium-balance period was smaller than during the following calcium-positive period. The same data show that in the well-fed milk cow these negative calcium balances are not due to a deficient calcium intake. So consistently are these negative balances encountered at the beginning of the lactation period that Lintzel[73] speaks of their occurrence as a physiological process.

From this viewpoint, the skeleton of the cow must not be considered merely as an organ of the body that has a certain mechanical function to perform, namely, to form the framework for the support of the remaining organs of the body, but it also constitutes a storehouse for the minerals from whence a certain amount may, in time of need, be withdrawn without seriously interfering with the health of the animal, much as the animal may store its fat and later use it in time of stress. The amount of minerals that can be withdrawn and the time during which such withdrawal may continue will depend upon the state of fullness of the storehouse. Likewise will this state of fullness also govern the quantity of minerals that can be stored daily under optimum conditions. Aside from the negative calcium balance encountered in the early portion of the lactating period, the behavior of the calcium and phosphorus balances of a lactating animal do not differ materially from those of a nonlactating animal, provided the mineral needs of the animal are fully met.

DETERMINATION OF CALCIUM AND PHOSPHORUS REQUIREMENTS

Since the whole process of calcium and phosphorus metabolism is a complicated one and factors enter into it that are as yet

not fully understood, it is difficult to determine the precise amount of the different mineral elements required for the most economical production of meat, milk and wool or for the maintenance of perfect health. However, certain broad principles have been established which may be used as a guide in arriving at the approximate amount that must be supplied in the feed of the different classes of animals. Difficulty is encountered in these determinations because it is not known just what portion of the minerals is assimilated by the animal, and what portion of them again leaves the body with the droppings without ever having become a part of the body tissues or fluids.

Much has been written regarding the percentage of calcium and phosphorus retained by the animal from the feed, but in calculating these, due regard has not always been given to the absolute amount of the minerals contained in the ration. It has been shown repeatedly that the animal body will utilize calcium and phosphorus more economically when supplied in amounts below or near maintenance requirements than when supplied at a higher level. It may happen, therefore, that on a certain level of calcium intake from a timothy hay ration the percentage of calcium assimilation may be in the neighborhood of 40 per cent, whereas when alfalfa hay is substituted for timothy hay, or calcium carbonate is added, thus almost doubling the absolute amount of calcium intake, the percentage assimilation may be only half that on the former ration. But on the whole it may be accepted that one fifth to one half of the calcium in the ration may be assimilated by the animal. In the case of phosphorus these figures run a little higher.

It is, however, highly important that the figures for maintenance be approximated as nearly as possible, for unless we know the calcium and phosphorus requirements for health, it is impossible to intelligently judge diseased conditions that may have developed as a result of improper or insufficient mineral supply in the feed. To obtain these figures has been the aim of numerous experiments, the first of which were probably carried out by Lehmann in 1858, and since carried forward by Soxhlet, Weiske, Kellner, Fingerling, Forbes, Theiler and coworkers, and others. The majority of these findings have been based upon short-term feeding experiments in which the amount of calcium and phosphorus retained by the animal was accepted as covering its maintenance requirements plus the amount utilized for the production of body gain.

For milk production one must consider that one pound of milk contains approximately 0.583 Gm. (9 gr.) of calcium and 0.297 Gm. (4.6 gr.) of phosphorus. The amount allowed in the feed for milk production must, of course, be increased above this quantity, depending upon the calcium and phosphorus utilization of the ration. Thus, Kellner[16] has accepted the amount of calcium and phosphorus required in the ration per liter (1.05 qts.) of milk production as 0.87 Gm. (13.4 gr.) of calcium and 0.7 Gm. (10.8 gr.) of phosphorus.

Additional figures for the mineral requirements are available from the work of others. Forbes *et al*[17] found 16 Gm. (0.5 oz.) of calcium and 10 Gm. (2.5 dr.) of phosphorus adequate for a 455-kg. (1,000-lb.) Angus steer. Theiler *et al*,[18,19] feeding 26.4 Gm. (0.9 oz.) of calcium and 12.2 Gm. (3 dr.) of phosphorus to a cow from 9 months of age to 36 months of age, during which time the cow increased in weight from 159 to 551 kg. (350 to 1,213 lbs.) and also calved, found this amount adequate, as indicated by the weight increase of the animal, the blood inorganic phosphorus and the histological appearance of the bone.

Theiler, Du Toit and Malan[20] and Otto[21] fed 22.92 Gm. (.75 oz.) of calcium and 11 Gm. (.35 oz.) of phosphorus to each of two high grade Friesland heifers for 26 months, beginning at 15 months of age. They grew from 273 kg. (600 lbs.) to over 455 kg. (1,000 lbs.) and had normal bones, as determined by histological examination and ash determination and normal blood-phosphorus content. It is interesting to note, however, that in the same experiment two other similar animals each received 4.5 Gm. (69 gr.) of calcium and 11 Gm. (2.8 dr.) of phosphorus. One had normal bones

TABLE I—Approximate food calcium and phosphorus requirements for growth and maintenance (in grams) for cattle over 15 months of age, per 455 kg. (1,000 lbs.) of live weight.

AUTHOR	CALCIUM	PHOSPHORUS	REMARKS
Kellner [16]	32.5	10.0	On basis of Henneberg's figures
Forbes *et al*[17]	16.0	10.0	Angus steer, in calcium and phosphorus equilibrium
Theiler *et al*[18, 19]	26.4	12.0	Friesland heifer from 9 months of age through gestation. Normal bones histologically
Theiler *et al*[20]	22.9	11.0	Friesland heifers from 15 to 41 months of age; no gestation. Bones normal histologically
Huffman *et al*[22]	—	10 to 11	Holstein heifers from 18 months of age through gestation
Otto[21]	16.36	8.34	Normally growing, healthy Friesland steer, from 18 to 44 months of age. Normal bones histologically
Theiler *et al*[20]	5.86	10.5	Friesland heifer from 20 to 42 months of age, through gestation. Marked atrophy of bones histologically
Theiler *et al*[20]	4.5	11.0	Friesland heifer 15 to 43 months of age. Marked atrophy of bones histologically

after 20 months on the ration, while the other showed a marked atrophy of the bone upon histological examination after 28 months on the ration and, chemically, a lower ash content, especially in the ribs (11 per cent decrease compared with the adequately fed animal).

In another experiment Theiler *et al*[18,19] fed two Friesland heifers weighing 220 and 213.6 kg. (483 and 470 lbs.), respectively, for 20 months on a ration containing 5.86 Gm. (90.2 gr.) of calcium and 10.5 Gm. (2.7 dr.) of phosphorus. According to all standards the phosphorus supply was adequate. The two heifers attained a weight of 550 and 518 kg. (1,210 and 1,140 lbs.), respectively, after being on the experiment for 22 months, and both calved. One had twins and soon thereafter became a bone chewer, although her inorganic blood phosphorus remained normal. The other did not become a bone chewer but microscopic examination of the bones at the end of the experiment showed marked atrophy of the bone, although nothing indicating rickets. Thus, in this animal 5.86 Gm. of calcium was inadequate.

The same authors, working with Friesland steers, fed one animal 15.7 Gm. (4 dr.) of calcium and 7.86 Gm. (2 dr.) of phosphorus, beginning at the age of 18 months and continued for 26 months. They found these amounts to be adequate, as indicated by histological bone examination, bone ash and inorganic blood phosphorus.

Huffman, Duncan, Robinson and Lamb[22] placed two groups of seven grade Holstein heifers on experiment at 90 days of age and carried them for five years, one receiving a low phosphorus ration and the other a high phosphorus ration. The calcium intake for the low phosphorus group ranged from 24 to 38 Gm. (0.8 to 1.25 oz.) in one sub-group and from 24 to 63 Gm. in another sub-group up to first calving. In the high phosphorus group the calcium intake ranged from 45 to 100 Gm. and above in one sub-group and from 42 to 69 Gm. in another. The phosphorus intake in the low phosphorus group ranged from 5.2 to about 11 Gm. and in the high phosphorus group from 10.3 to about 30 Gm. daily, all depending upon the gain in weight or approach of the end of first gestation. The authors conclude that 10 to 12 Gm. of phosphorus daily was adequate for maintenance, normal growth and development of a fetus from 18 months of age to first calving, as indicated by blood phosphorus and strength and size of calf at birth. For milk production the authors suggest 0.75 Gm. (11.5 gr.) of food phosphorus above maintenance per pound of milk produced daily. Their results indicate that not less than 17 Gm. of phosphorus should be fed daily during low production and during the dry period when the cow is in an advanced stage of gestation. The findings are summarized in table I.

The figures of Theiler and his coworkers[18,19,20] do not give the exact amount for maintenance upon a progressing weight basis because all of their animals were put

on a uniform amount during the whole time of the experiment. But the live weights of Otto's[21] steer are given, from which the daily gain can be calculated and, therewith, the amount of calcium and phosphorus remaining for maintenance. This gain amounted to one-half pound per day in the eleven months during the middle of the experiment. Since this part of the experiment probably represents the crucial time in the mineral storage of the animal and also the least fat production, it may be taken as a fair average for maintenance.

TABLE II—Approximate calcium and phosphorus requirements (in grams) per 45.5 kg. (100 lbs.) of live weight for young, growing animals.

Author	Calves		Yearlings		2-Yr.-Olds		Remarks
	Ca	P	Ca	P	Ca	P	
Archibald and Bennett[23]	3.85	1.8	3.27	1.7	3.7	1.2	Did well
Lindsey, Archibald and Nelson[74]	4.46	2.79	2.89	2.26	2.59	1.8	Did well
Henderson and Weakley[24]	6.3	5.2	3.7	4.0	3.0	1.2	Developed rickets
	12.9	4.8	11.3	3.4	8.5	1.2	Developed rickets
	7.1	4.7	3.8	3.5	2.1	2.3	Normal
Huffman *et al*[22]	10.0	2.2 to 1.8	6.1	1.8 to 1.4			Phosphorus deficiency; low blood phosphorus to 18 months of age

During these eleven months the weight of the steer increased from 409 to 484 kg. (900 to 1,065 lbs.) and, thus, the animal had a mean weight of 452.4 kg. (996 lbs.). On this basis one may accept the amounts fed as fully meeting the requirements of a 455-kg. (1,000-lb.) animal, especially since during the next eight months the animal had a further increase in weight of 93.2 kg. (205 lbs.).

These findings give us an approximate idea of the calcium and phosphorus requirements of animals over 15 months of age. For these requirements, in the case of growing calves, we may compare the results given in table II, remembering, of course, that the rapidly growing young animal requires a relatively larger amount of these elements per pound of body weight than a more mature animal. In these cases the calcium intake was comparatively high but the phosphorus intake in some instances was on the borderline or inadequate and, for that reason, is of particular interest.

Rôle of Vitamin D in Mineral Metabolism

It is an established fact that vitamin D is active in the regulation of the mineral metabolism and, especially, in the utilization of calcium and phosphorus. It has been shown that it increases the utilization of calcium and phosphorus and is most helpful in those cases where the supply of these two elements is low or inadequate or where there is an imbalance between them. Thus, Shohl and Wolbach[25] have shown that in the absence of vitamin D, rickets can be produced in rats receiving high-calcium low-phosphorus diets, or low-calcium high-phosphorus diets, or low-calcium low-phosphorus diets, which implies that rickets can be produced with any ratio of calcium to phosphorus. These results were established on young, rapidly growing rats and hold true for that class of animals, but they probably do not hold true for mature rats.

While it is true that rickets can readily be produced in young, growing calves in the absence of vitamin D or rations with a low vitamin D supply (Bechdel *et al*[26,27], Rupel *et al*[28], Bechtel *et al*[29]) and adequate amounts of calcium and phosphorus and favorable calcium to phosphorus ratio, yet in nature vitamin D is so abundant and so widely distributed that under practical methods of animal husbandry it can not

easily be evaded. Thus, the exposure of the animal to direct sunshine for only a few hours a day will probably give it all the antirachitic factor that it needs. Furthermore, but few roughages are in common use that do not contain this factor in sufficient amount for the ordinary needs of the animal, except probably under prolonged storage. A combination of sunshine and sun-cured hay as roughage ordinarily does not fail to supply this factor in abundance.

An instance can be cited, however, where under practical dairying conditions in the South, calves were exposed to the hot summer sun practically all day while running on an excellent green Bermuda grass pasture and fed an excellent grain ration together with some hay for additional roughage, yet they developed rickets. The professor of dairy husbandry could not believe that the ration was at fault, but when the calcium to phosphorus ratio was changed from 1 to 6 in the ration to one of 2 to 1 by the withdrawal of bone meal from the ration and the substitution of calcium carbonate in the proper amount, the trouble was soon eliminated.

Another interesting case is the following. The time was the middle of January, with cloudy weather practically all day. Long yearling calves were being fed cottonseed cake to be fattened for market on pasture in which the animals could partake of an abundance of tall prairie grass dried on the stem. No other feed was supplied. When the young animals almost had reached the stage where they were ready for market, they went off feed, went lame and lost weight. After ten days or two weeks, and after losing about 100 pounds in weight and all the profit for the owner, they came back on feed again. In an adjoining pasture, yearling lambs were being fed in the same way. The owner complained that a number of them had become paralyzed behind, and one specimen, kept in a sling made from an empty feed bag, was shown to the writer. Upon autopsy it was found that the spinal column was broken in the lumbar region and that all of the bones were so soft that they could be cut readily with a butcher knife. The owner was advised to add calcium carbonate to his cottonseed cake in some way so as to balance the excess phosphorus. When this was done, the trouble terminated.

The cause of rickets and osteomalacia in live stock has been known for a long time and has been attributed to an inadequate supply of minerals in the food. It is not the object of this paper to give an extensive review of the literature on the subject as far as it affects the practice of veterinary medicine, for such has been given by a number of authors. One of the earlier extensive treatises on the subject is that by Klimmer and Schmidt.[30] Their observations covered mostly farm animals kept usually under intensive agricultural conditions. The authors gave extensive observations on the calcium and phosphorus content of the feed and numerous analyses of bones of both healthy animals and animals suffering from osteomalacia.

At an earlier date (1884) Hutcheon in South Africa (Marston[31]), observing *lamsiekte* and *styfsiekte*, suspected a phosphorus deficiency as the cause, and as late as 1903, he was quite emphatic on this point. It fell to the lot of Theiler to continue the investigation of these two diseases and finally to establish their cause, with the result that his findings were the signal for an exhaustive investigation of the mineral requirements of our farm animals.

Theiler[32] definitely established a phosphorus deficiency as the cause of *styfsiekte* and applied the term aphosphorosis to the condition of phosphorus deficiency. Such aphosphorosis had been known for a long time under various local names, such as peg-leg, cripples, creeps, sweeny, stiffs, and possibly others, and investigations to elucidate the actual cause of these conditions, soon after it became known that a phosphorus deficiency in our farm animals may occur under range conditions, brought to light not only the vast extent to which aphosphorosis existed but also an intimate relationship between aphosphorosis and the

economical production of domestic animals under range conditions.

APHOSPHOROSIS AND "LOIN DISEASE" OF CATTLE

The writer[33,34] has had an opportunity to devote much time to the study of this condition in connection with his studies on so-called loin disease of cattle, an intoxication occurring in cattle along the gulf-coast region of Texas. In these investigations under pasture conditions covering a period of six years with an average of 150 cows, experiments were carried on to control the consumption of putrid carcass material by feeding the cows phosphorus supplements in various forms. It was desirable to control the consumption of putrid carcass material because it is sometimes toxic, and when such material was consumed by the cow, she became more or less paralyzed and died, a condition locally known as loin disease.

The country in which this aphosphorosis occurs along the gulf-coast region affords a luxuriant growth of grasses, but they are rather low in phosphorus. Many samples of more or less mature grass that the writer analyzed from the particular pastures under study averaged 0.12 per cent P_2O_5 and 0.64 per cent CaO on an air-dry basis. The protein content ranged from a low of 4.5 per cent to a high of 7 per cent under favorable conditions, with a majority of the samples running between 5.5 to 6.5 per cent protein. Digestion experiments (Fraps[35]) show 27.8 per cent digestible protein. If one allows a consumption of 8,000 Gm. (15.6 lbs.) of air-dry grass, a cow would receive 4.2 Gm. (64.7 gr.) of phosphorus daily. These figures are a little low, but during the favorable season, when the grass is more or less green and not fully matured, the cattle do well on it. On the whole, however, top production both in meat and offspring is not attained on the grass alone, as will be seen from the data to follow.

RELATION OF PHOSPHORUS DEFICIENCY TO BONE-CHEWING HABIT OF CATTLE

The bone-chewing habit of many of the cattle in the area under consideration was taken as an indication of a phosphorus deficiency and, accordingly, phosphorus was supplied in the form of bone meal mixed with sodium chloride which was fed in individual boxes every day in the year. The experiments were continued over a period of six years and a record was kept of each animal on the amount of bone meal consumed, the bone-chewing habit, loss or gain in weight, reproduction, and the amount of supplemental feed required in the latter part of the winter.

The cattle were assigned to five different pastures for the purpose of trying out different phosphorus supplements and different proportions of bone meal and salt. Only the results obtained with bone meal will be discussed here. Each pasture contained an equal number of animals which were not fed and served as controls to those that received bone meal. From the second year on, yearling offspring from the cows on experiment became available; these also were divided equally, one half placed on bone meal and salt mixtures, and a record kept of the performance of all.

EFFECTS OF BONE-MEAL FEEDING ON WEIGHT, BONE-CHEWING HABIT AND REPRODUCTION

Bone-meal feeding was started in the middle of June 1924, and the beneficial effects of feeding bone meal soon became apparent even in 5-year-old cows. They put on more flesh and looked better than the control cows and those which were bone chewers stopped this habit. The following year, the first full year in which the cattle were carried through all four seasons of the year, yielded interesting observations that also could be made in each succeeding year. Fresh young grass makes its first appearance in this area about the middle of March, and by the first of April it is already abundant enough to afford fair picking. As soon as sufficient young grass became available to the cattle, all of those on bone meal stopped eating bone meal and continued to refuse it until the grass became a little tough, about six weeks to two months later.

A second phenomenon ran hand in hand with this observation, namely, the bone-

chewing habit of the control cows. It was observed that as fall approached during the previous year and during the winter, practically all cows stopped the bone-chewing habit. During the following spring when the bone-meal cows came back to eat bone meal again, after having stopped eating bone meal after the appearance of green grass, it was interesting to observe that the control cows also started to chew bones again.

And still a third phenomenon bearing on the question of phosphorus deficiency was observed. The cows were weighed once a month. These weights showed that the body weight of the cattle was at its lowest ebb at the time green grass appeared and that it continued to increase month by month until about the first of November of each year, when it started to decrease to reach its low ebb sometime in February or early March. This loss in weight sometimes amounted to as much as 73 kg. (160 lbs.) for a cow weighing 330 kg. (725 lbs.) on November 1, and averaged 54 kg. (119 lbs.) for 32 cows subsisting entirely on range vegetation throughout the year. The interesting observation could be made each year that beginning about the time that the increase in body weight ceased in the fall, the bone-chewing habit also ceased and, as soon as the body weight began to increase in the spring, the bone-chewing habit would be resumed (graph 1).

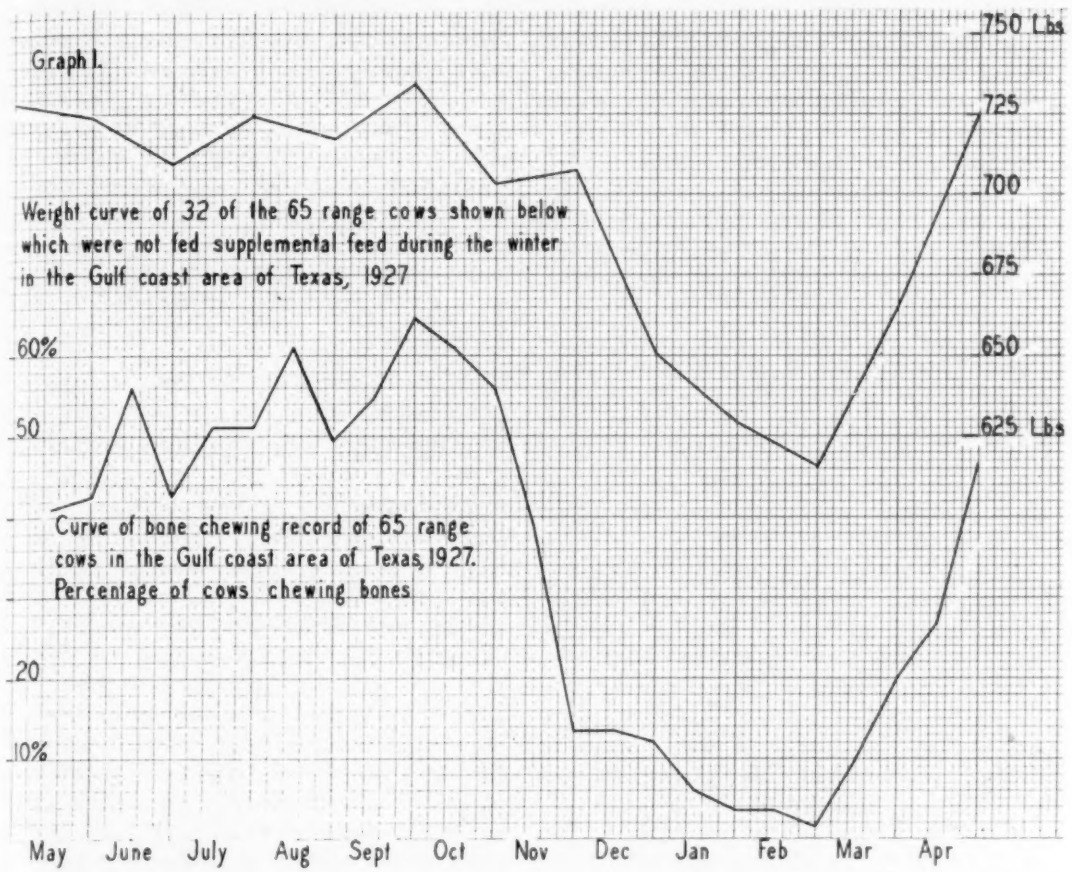
Thus, there appears a close correlation between the phosphorus supply in the feed and the gain or loss in body weight as far as the bone-chewing habit can be accepted as an indication of a phosphorus deficiency in the animal. As a general rule this may be accepted, but it does not hold true in each individual case. The author has made numerous observations where cows suffered from acute aphosphorosis and, yet, could never be observed to chew bones.

A difference in reproduction between the two groups of animals also was observed. This difference was apparent in each year so that at the end of six years the average calf crop for the cows fed bone meal was 11 per cent higher than for the control cows—64 per cent for the cows on bone meal and 53 per cent for the control cows. The writer attributes this to a phosphorus deficiency in the soil. The phosphorus deficiency of the soil leads to a phosphorus deficiency in the plant and a low protein content of the same. This low protein content in the vegetation of the area under discussion is near the upper limit of protein content of the ration, which, according to Guilbert and Goss,[36] does not support normal reproduction in the rat. The protein content is not only low; it is likely that it is also biologically inferior and that if adequate amounts of phosphorus were available from the soil, it would have a higher phosphorus content and also a higher and biologically more complete protein. Be that as it may, the fact remains that the feeding of bone meal furnished the animals additional phosphorus in the very low supply that they were getting from the native vegetation, and the reaction was a greater calf crop while they were otherwise on the same diet as the control animals. Thus, the action of all other factors that might complicate the problem, according to Eckles and his coworkers,[37] can be regarded as having been excluded.

On the basis of the grass analyses given above, the calcium intake on the native vegetation was about 36.5 Gm. (1.2 oz.) daily, an amount far beyond the needs of the animal, aside from the fact that Theiler,[38] Fitch and his coworkers[39] and Palmer *et al*[40] showed, in contradiction of the results obtained by Hart and his coworkers,[41] that a calcium deficiency does not affect reproduction in cattle.

There was not only an increase in the calf crop but the cows getting bone meal raised better calves. Thus, during four consecutive years for which these figures happen to be analyzed, the 97 calves of the cows getting bone meal averaged 9.55 kg. (21 lbs.) more at 200 days of age when they were weaned than the 89 calves of the control cows. That the heavier calves from the cows getting bone meal also commanded a better price per pound on the market need hardly be pointed out.

A striking difference in the growth and development in steers when placed on bone



meal as yearlings also was observed. A similar condition prevailed in heifers also, but it is not easy to follow the advantages of additional phosphorus feeding in these when growth and gain in weight are taken as the index because of the early interference of reproduction. Two groups of three steers each were included in a test of which the control steers averaged 6.8 kg. (15 lbs.) more when they were started as yearlings. After 16 months the three steers getting bone meal averaged 46 kg. (101 lbs.) more than the three control steers and maintained this advantage during the following two years.

Another perceptible fact that stood out rather prominently in these experiments was the higher death rate in the control animals than in the cows on bone meal. At some time during each year an attempt was made to equalize the number of animals in each group by purchasing new animals to take the place of those which died. In order to keep the two groups equalized, 62 cows were bought during the first four of the six years to be placed in the bone meal group, of which 39 survived the full four years. During the same time 104 cows were bought for the control group, of which 42 survived during the same time, thus giving a death loss of 23, or 37 per cent, in the group on bone meal and 62, or 59.5 per cent, in the control group. When these results are viewed in their entirety, they will be found to be similar to those obtained by Theiler *et al*[42] in South Africa.

Manifestations of Aphosphorosis

The question is now in order, how does aphosphorosis manifest itself? Some of these manifestations are already apparent from what has been said above. In these considerations the disadvantages to a more

or less mature cow subsisting solely on a phosphorus-deficient vegetation have been brought out, but the fact that some cows may suffer a breakdown under the severe strain of trying to garner sufficient nourishment from such deficient vegetation for their own maintenance and to produce offspring at the same time, needs further consideration. When this strain becomes too severe, the first thing that one usually observes is a loss in body weight. This loss in weight is the only symptom in the early stage of the disease in the cow and is soon followed by a peculiar stiff gait, the animal usually taking shorter strides than normally. When such an emaciated cow walks along with this peculiar stiff gait, it appears that she is just creeping along; hence the local designation "creeps."

The continued loss in weight in this condition must be attributed to a direct inadequacy of phosphorus with which to carry on, since food utilization is not decreased in a state of aphosphorosis, as shown by several authors (Kleiber, Goss and Guilbert[43]; Henderson and Weakley[24]; Riddell, Hughes and Fitch[44]; Huffman *et al*[13]). Food consumption is markedly decreased so that the cow grazes but little and soon stops altogether, spending much of her time lying down in order to rest her aching bones. In this state she is an emaciated, miserable looking wretch and, unless help is offered, death will follow. Naturally the calf can not thrive when its mother is in such a condition and it will soon lose its bloom, will lag in its development, become poor and, unless it already has attained the age where it can subsist upon the grass, also may perish.

To cope successfully with a phosphorus deficiency is more or less an individual problem. At least, we would reach this conclusion from the observation that some cows develop creeps each year they have a calf while other similar cows, running on the same range and subjected to identical conditions in every way, show no signs of creeps.

A young, growing animal needs relatively more phosphorus for maintenance and growth than a mature animal. It is for this reason that symptoms of aphosphorosis were so frequently observed in our young, growing stock, especially from sometime after weaning until the animals approached 3 years of age. They manifest the same general symptoms as the more mature cow and, in addition, enlargements around the lower end of the metacarpus and metatarsus frequently are observed. During especially unfavorable years it is not unusual to find such enlargement of the bones on over 50 per cent of all the 2- and 3-year-olds.

CASES OF CALCIUM-PHOSPHORUS IMBALANCE

In this paper consideration has thus far been given only to aphosphorosis on the range where the animal subsists on a vegetation that usually contains not only more calcium than phosphorus but also adequate amounts of calcium so that a simple phosphorus deficiency exists. The animal feeds itself, so to speak. When, however, man undertakes to feed the animal, then conditions become more complicated, for man usually reaches for concentrates in which a large amount of phosphorus is contained in a very small package. The result is that, in his zeal to be kind to the animal or to increase its production to a maximum capacity, he will sometimes overdo matters. The concentrates are much higher in phosphorus than in calcium and, unless care is taken to adjust this difference, the animal is liable to suffer from a calcium deficiency. Such a condition is reported by Becker and his coworkers[45] from Florida, where a vegetation growing on gray, sandy soils is rather low in calcium. When hay grown on such soils was the main source of the roughage used in the dairy herd, it was observed that many of the cows at one time or another had broken bones. A check on the rations used showed that the cows were getting only 40 per cent of the calcium and four times the amount of phosphorus called for in Wellmann's[46] standard. It is remarkable that at no time did the cows show any ill effects except the fragility of bones. They ate well and remained quite fat.

On the other hand, Theiler and his coworkers[20] showed that, with an adequate

phosphorus intake, an increase or a decrease of calcium over or below an adequate intake of calcium decreased the phosphorus retention considerably, and a bone analysis verified the indication of a poorer utilization of minerals. Since the phosphorus intake remained the same in both cases, it would seem that the calcium to phosphorus ratio must be held responsible for this phenomenon.

The deleterious effect of excessive calcium intake coupled with a phosphorus intake slightly below an adequate amount also was observed by Eckles and his coworkers.[47] When feeding in the neighborhood of 175 Gm. (5.8 oz.) of calcium to about 9.5 Gm. of phosphorus per 455 kg. (1,000 lbs.) of live weight, his cow developed all of the symptoms of aphosphorosis and a low inorganic blood phosphorus.

Marek *et al*[48] reported a case on a farm where fractures of bones among the dairy cows were quite frequent. During the four months preceding their findings no fewer than 20 cows had to be slaughtered because of bone fractures. Their investigation showed that the protein in the ration had been calculated on a basis of an increase of 14 liters (14.8 qts.) of milk and that this increased the calcium and phosphorus intake to 269.25 Gm. of calcium and 41.71 Gm. of phosphorus. The feed probably contained only traces of vitamin D. The particular cow examined exhibited all of the clinical symptoms of an aphosphorosis; the inorganic blood phosphorus was low and, histologically, large masses of osteoid tissue were found in the bones, which also showed a low ash content. In conformity with their previous views[49] the authors stressed the importance of the *Erdalkali-Alkalinität* $(CaO + MgO) - P_2O_5)$ and sought to explain the development of osteomalacia in this case on this basis. According to these authors, an *Erdalkali-Alkalinität* of +25 mg. equivalents per 100 Gm. of dry matter is to be considered as most advantageous, while one much below or much above is considered as detrimental. In the case in question the *Erdalkali-Alkalinität* amounted to +100.7 mg. equivalents. According to Kellner's[16] standard of mineral requirements in lactating cows, the phosphorus intake in this cow was more than adequate while the calcium intake was greatly in excess. The case shows definitely that osteomalacia also can be produced in ruminants on an alkalotic basis and not only on an acidotic diet, as was heretofore generally believed.

In further experiments Marek *et al*[50] fed calves a ration containing practically no vitamin D. The calcium intake was 6.07 and 7.59 Gm. and the phosphorus intake 9.69 and 10 Gm., respectively, in the two calves. The *Erdalkali-Alkalinität* was —10.77 mg. equivalents in the first calf and —16.25 mg. equivalents in the second. In both animals the ash content of the bones was decreased and, histologically, showed marked rachitic changes. The inorganic blood phosphorus was increased rather than decreased. The authors concluded that this experiment furnishes proof that rickets in ruminants can be produced with a ration containing an excess of phosphorus or a negative *Erdalkali-Alkalinität*.

OSTEODYSTROPHIA FIBROSA (=SOFTENING OF THE BONES) IN HORSES

A diseased condition in horses manifesting itself in a softening of the bones has been known for a long time and is recorded in the literature under various names, such as osteoporosis, osteomalacia, ostitis fibrosa and, more recently, osteodystrophia fibrosa. An interesting account of a widespread occurrence of it among British army horses and mules in South Africa, and also among civilian horses and mules, was published by Captain A. H. Lane[51] in 1906. At that time the cause of the disease was not known. Some believed it to be of microbic origin while others attributed it to a deficiency of lime salts and phosphoric acid in the food. Captain Lane leaned toward the latter theory. He had the opportunity to carry on some actual feeding trials in which the ration being fed to affected horses was changed to good oats, wheat bran and alfalfa hay, which resulted in the disappearance of the disease.

In the following year Theiler[52] as veterinarian and Ingle[53] as chemist published their respective findings as separate docu-

TABLE III—Relation of daily intake (in grams) of calcium and phosphorus in horses per animal to osteodystrophia fibrosa.

AUTHOR	CA	P	RATIO CA/P	TIME REQUIRED FOR SYMPTOMS TO DEVELOP
Scheunert and Schattke[54]	14.3	25.76	0.54	Osteomalacia developed
Niimi[56] (used old horses)	1.49	12.04	0.124	Osteomalacia in 5 months
Niimi and Aoki[57]	2.58	20.69	0.124	Osteomalacia in 4 months
	20.0	18.16	1.1	None developed in 8 months
Kintner and Holt[58]	13.0	23.14	0.56	Osteomalacia in 9 months
(Used army horses)	11.21	12.7	0.91	None developed in 9 months
Sturgess and Crawford [59]	20.11	62.5	0.324	More than 9 months
	13.6	56.37	0.242	More than 9 months
	13.6	16.35	0.832	None developed
Lane,[51] estimated from ration	4.88	19.16	0.254	Produced osteomalacia
	176.1	52.5	3.35	Cured osteomalacia
Groenewald[60] (yearlings)	3.4	23.7	0.143	Osteofibrosis in 9 months
	3.23	12.4	0.26	Osteofibrosis in 9 months
	37.2	12.4	3.0	None developed in 2 years
Marek, Wellmann and Urbanyi[48]	11.16	44.7	0.25	Rickets in 6 months
(Colts, 8 months old)	47.5	18.0	2.64	None developed in 11 months

ments in a coöperative undertaking to solve the cause of this malady, which was raising havoc with the horse and mule population of South Africa. Theiler, on the basis of epizoötiology, insisted that the disease must be of microbic origin, but not only failed to find any microbes but also failed to prove that it was contracted by cohabitation. Ingle, on the other hand, on the basis of an analysis of bones from both diseased and healthy horses and also of feed analyses, ventured the theory that "the abnormal conditions of the bones are favored by the use of foods not necessarily deficient in lime or phosphorus but in which the ratio of lime (and perhaps magnesia) to the phosphoric acid is too low."

AN OUTBREAK OF OSTITIS FIBROSA IN GERMAN ARMY HORSES

Only a few years later Scheunert and Schattke[54] encountered the disease in army horses in Germany and, after a thorough survey of the feeding practices and feed analysis, attributed it to the low calcium intake with the ration. A most interesting sidelight was cast upon the etiology of this disease by a destructive outbreak of a disease among the horses of a certain army regiment in Germany which manifested itself by a softening of the bones. Joest and Zumpke[55] described it as ostitis fibrosa because the histological changes in the bones clearly did not correspond to those found in osteomalacia of humans or animals.

It fell to the lot of Scheunert and his coworkers[61,62,63] to investigate the etiology of the outbreak. They found that the mineral metabolism was taking a normal course, as far as could be detected by chemical analyses, and that the feed itself was not at fault, since horses fed from the same feed supply outside of the army stables did not contract the disease. Hereupon, attention was directed at the bacterial flora of the intestinal tract. It was found that this had undergone a complete change and consisted mostly of acid and phenol producers. The trouble was not overcome until a thorough cleaning and disinfection of the stable was undertaken. It appears from the literature that this is an isolated instance where a destructive outbreak of softening of the bones in the horse has been traced to a "dysbiosis" of the bacterial flora of the intestinal tract.

The experimental production of osteo-

TABLE IV—Behavior of osteomalacia in horses with respect to different amounts and ratios of calcium and phosphorus in ration, as reported by Kintner and Holt.[58]

No. of Horses	Amount Fed (Gm.)		Ratio Ca/P	Clinical Results
	Ca	P		
8*	10.14	18.8	0.539	Disease progressing
1*	12.14	18.8	0.645	No clinical improvement in 90 days
8*	35.0	18.8	1.86	Disease arrested in 90 days
5*	22.85	18.8	1.21	Definite improvement in 98 days
5*	24.28	15.72	1.54	Definite improvement in 98 days
5*	24.28	37.12	0.654	Disease progressing in 3 out of 5 in 98 days
31†	13.2	23.14	0.57	Three developed osteomalacia in 9 months
25†	12.6	12.71	1.0	No osteomalacia in 9 months

Status at outset: * = Osteomalacia; † = Clinically healthy.

dystrophia fibrosa was next studied by Niimi[56] and Niimi *et al*[57,64] in Japan, and by Sturgess and Crawford[59] in India. In both instances the disease was produced by feeding an excess of phosphorus over calcium with a wide calcium/phosphorus ratio. Clinically and pathologically the disease thus produced was identical with that observed elsewhere and left little doubt that an imbalance of calcium and phosphorus in the ration was the seat of the trouble. Additional evidence of this fact has been furnished more recently by the work of Kintner and Holt,[58] by Groenewald[60] and by Marek *et al*.[65]

Table III gives in a condensed form the salient facts concerning the amount of calcium and phosphorus fed and the calcium/phosphorus ratio under which the disease occurred or could be produced. Table IV gives additional figures on the finding of Kintner and Holt. In all instances the disease could be stopped by furnishing an adequate calcium supply in the ration and adjusting the calcium/phosphorus ratio to about 1 or a little above.

Calcium and Phosphorus Requirements of Horses

Under these conditions data on calcium and phosphorus requirements of the horse in health become extremely important. Unfortunately, but little information is contained in the literature on this point. Scheunert, Schattke and Weise[66] found calcium equilibrium in a horse receiving 11.7 Gm. (3 dr.) of calcium in the ration and phosphorus equilibrium when 18.2 Gm. (0.6 oz.) of this element was supplied. Mitchell and McClure[67] calculated from available published data that a 182- to 727-kg. (400- to 1,600-lb.) horse requires 13.5 Gm. (3.6 dr.) of calcium and 8.9 Gm. (2.4 dr.) of phosphorus for maintenance and growth. Further evidence as to the approximate requirements of these two mineral elements by the horse can be obtained from the feeding experiments as

TABLE V—Osteoporosis in the horse, as produced by Sturgess and Crawford.[59]

Pony No.	Daily Intake		Ratio Ca/P	Results
	Ca (Gm.)	P (Gm.)		
1	6.54	56.61	0.11	Osteoporosis after 9 months. Fell and broke back
2	13.68	56.61	0.24	Osteoporosis after 9 months
3	20.12	62.05	0.32	Osteoporosis beginning after 9 months
4	18.57	58.59	0.3	In good condition; no symptoms of osteoporosis at end of 14 months
5	4.85	53.3	0.09	Osteoporosis after 9 months; down after 10 months
6	13.5	16.15	0.84	In good condition; no symptoms of osteoporosis after 14 months
4*	5.0	38.0	0.13	To this ratio after 14 months; osteoporosis two months later

*Pony 4 was changed to this ration after being on ration as listed above for 14 months.

shown in table III, in those cases where the control animals did not develop osteomalacia. According to the figures furnished by Kintner and Holt, 12 Gm. (3 dr.) of calcium and 12.7 Gm. (3.2 dr.) of phosphorus, and those by Sturgess and Crawford, 13.6 Gm. of calcium and 16.35 Gm. of phosphorus, were adequate. Klimmer[68] recommends 35.7 Gm. of calcium and 10.66 Gm. of phosphorus for a horse weighing 500 kg. (1,100 lbs.). Although these figures are too limited to draw a definite conclusion as to the exact amounts of the two elements necessary, it seems that in round figures 12 Gm. (3 dr.) of calcium and phosphorus each are adequate.

Sturgess and Crawford fed six ponies on rations containing different amounts of calcium and phosphorus, as indicated in table V. Of the six ponies, No. 6 served as a control and remained healthy. For some unknown reason pony 4 did not develop the disease during 14 months on the ration, but when he was changed to a greater excess of phosphorus over calcium, he broke down two months later. It thus appears that at the time the change in ration was made he was in the incipient stage of the disease.

The severity of the clinical symptoms in the different ponies was in the following order: No. 5, 1, 2, 3, 4 and 6. Note that they correspond exactly to the ratio of Ca/P in the ration. Since the absolute amounts of calcium fed to the different ponies varied greatly, no definite conclusions can be drawn from the experiment as to the exact rôle of inadequate amount of this element in the causation of osteomalacia. Pony 4 did not show symptoms after 14 months on the ration, although No. 3, getting practically the same ratio of Ca/P and, in the light of our present knowledge, probably an adequate amount of calcium, showed symptoms at the end of nine months. However, when the ration of pony 4 was changed at the end of 14 months to a calcium intake of 5 Gm. (1.3 dr.) and a phosphorus intake of 38 Gm. (1.3 oz.), he showed the first signs of osteoporosis after two months on this new ration and a fracture of the spinal column a month later.

Since two months on the new diet is hardly sufficient time to cause such a severe drain on the mineral reserves as to exhaust them to the point where bone enlargements occur, it may be assumed that pony 4 had already suffered a severe drain of his calcium reserve at the end of 14 months on the ration and might ultimately have broken down on it. Evidence of osteomalacia appeared even in a shorter time in the experiments of Niimi. Reference to table III will show that he not only fed a wide Ca/P ratio but also a low absolute amount of calcium and that his horses came down in about one half the time as those of Sturgess and Crawford.

As indicated above, the excess phosphorus, in being eliminated from the body, carries with it a large portion of the calcium reserve of the bones. That this is possible was clearly shown by Scheunert and his coworkers[66] in the horse. They found large amounts of calcium reserve being excreted from the body when excess phosphorus was fed. This causes a weakening in the structure of the bone and, ultimately, the symptoms manifested by the animal.

Theiler[69] regarded osteodystrophia fibrosa as a disease distinct from osteomalacia and rickets. Marek *et al*,[65] however, recently showed in their experiments that rickets can be produced in colts with a diet having an excess of phosphorus over an adequate calcium content, and they believe that osteodystrophia fibrosa develops on the basis of pure rachitic change. They fed a colt 11.16 Gm. (3 dr.) of calcium and 44.7 Gm. (1.5 oz.) of phosphorus daily, which gives a Ca/P ratio of 0.25 over a period of eleven months. The control animal receiving 47.5 Gm. of calcium and 18 Gm. of phosphorus for the same period remained healthy.

MANIFESTATIONS OF OSTEODYSTROPHIA FIBROSA

In the incipient stage the horse shows no definite changes and the symptoms presented are only variations in the activity of the animal. One may observe that the animal is less active, spends more time

lying down and soon may develop more or less stiffness. Stumbling may become apparent and a peculiar shortening of the stride develops. Lameness also develops and the animal often shifts its weight from one foot to the other. As time goes on, lameness increases, the horse moves with a stiff, stilted gait and is loath to trot, but if forced to do so shows a peculiar tucked-up abdomen. It is quite evident that its movements are painful.

An examination of the bones of the head may reveal slight bilateral enlargements of the bones of the face or the rami of the mandibles. At first these are only slight but, unless the cause is removed, the enlargements will increase in size until they are quite prominent. In the maxilla the enlargement occurs at a point directly above the roots of the upper molars. In the mandible the first bony enlargement may be noticed as a small, barely preceptible ridge along the side of the roots of the lower molars. As time elapses, however, the enlargement may involve practically all of the mandible, but especially the horizontal branches of the same.

Histologically these enlargements show a complete reconstruction in the architecture of the bone in which the animal attempts to conserve the strength of the bone with the small absolute amounts of calcium now available. In doing so, it must sacrifice the rigidity and increase the volume of the bone. This lack of rigidity does not only manifest itself in enlargements but also in other ways. Thus, it not infrequently happens that ligaments are torn loose or that the animal fractures a limb or its back upon a simple fall or a sudden strain.

The fracture of the head of the femur occurring in Marek's experimental animal and the large number of such cases in young horses reported by Hellmich[70] were the result of osteodystrophia fibrosa. The animal shows a lack of appetite, gradually becomes poor, emaciated and shows a rough, stary coat. Blood analysis may show a decrease in the calcium content but the phosphorus content is not changed significantly. Chemical analysis of the bones shows a significant decrease in ash content in which no significant change in the ratio of calcium to phosphorus is found, but there is a marked decrease in the amount of magnesium present (Marek *et al*,[65] McCrudden[71]). Grossly the bones are softer and weaker than normal and histologically show much uncalcified osteoid tissue and enormous quantities of fibrotic tissue which has replaced the original bone tissue.

PRINCIPLES OF TREATMENT AND PREVENTION OF MINERAL DEFICIENCIES IN HORSES AND CATTLE

The basic cause of the trouble being a deficiency, treatment and prevention must be identical. The remedy lies in supplying the needed mineral elements in the proper amounts and proportion. Rations compounded alike as regards kind and amount of feedstuff may or may not supply calcium and phosphorus in the same or like amounts, depending upon the locality where the feedstuffs were grown and the weather conditions under which they were grown and harvested. It may easily happen that in one year a ration may lead to the development of a mineral deficiency in the animals while the next year it may not do so because of the possible variation from year to year in the mineral components of the ration. The time during which the feeding of the ration is continued also must be given careful consideration, as well as the age of the animal and its probable mineral storage. The consideration of all these factors gives opportunity for the exercise of good judgment and the possibility of error. It is advisable, therefore, to supply these elements in amounts above minimum requirements rather than to figure on bare maintenance requirements. This is especially true when a chemical analysis of the feedstuff in question is not at hand. The livestock owner who is conversant with the principles of animal nutrition is not likely to fall into the error of an inadequate supply of calcium and phosphorus for his animals or an improper balance of them, but if he should, once the error is pointed out to him, he is not likely to repeat.

In the horse, when the lower limits of requirements are approached, it is almost imperative that calcium and phosphorus be supplied in approximately equal amounts if thrifty growth and development and continued good health are to be maintained, while the cow is capable of coping successfully with a much wider ratio of calcium to phosphorus. For this animal the calcium content may be double that of the phosphorus or the phosphorus double that of the calcium, provided the absolute quantity of the element fed in the lesser amount fully meets the requirements of the animal. The levels below which it is not safe to allow the absolute amount of calcium and phosphorus to fall are apparent from the data cited above. For the growing bovine it should not fall below 2 Gm. (31 gr.) of phosphorus and 3 Gm. (46 gr.) of calcium per 45.5 kg. (100 lbs.) of body weight; and for cattle approaching maturity, 10 Gm. (2.5 dr.) of phosphorus and 15 Gm. (0.5 oz.) of calcium per 455 kg. (1,000 lbs.) of body weight, or about one half that for the young, growing animal. For the lactating cow due allowance must be made on the basis of milk produced. The amount required in the ration, according to Kellner's[16] standard, is 0.87 Gm. (13.4 gr.) of calcium and 0.7 Gm. (10.8 gr.) of phosphorus per liter (1.05 qts.) of milk produced.

For the horse 12 Gm. (3 dr.) of calcium and phosphorus each in the feed for 1,000 lbs. of live weight is adequate to prevent the development of a deficiency. One must keep in mind in this animal the importance of a rather narrow ratio of calcium to phosphorus. The experiments of Groenewald[60] and of Marek[72] and his coworkers show that with 12 Gm. of feed phosphorus, 2½ to three times that amount of calcium may be fed without producing the disease in two years. These latter amounts coincide with those recommended by Klimmer.[68] This indicates that also the horse can tolerate a fairly wide ratio of calcium to phosphorus when the absolute amounts fed are adequate.

The amounts shown above indicate the lower limits of safety rather than the optimal amounts. It is well known that these amounts may and frequently are greatly exceeded without deleterious effect upon the health of the animal as long as both elements are fed in adequate amount and reasonably approach the ratio recognized as the most optimal. This ratio is most likely to be too wide in grain-fed animals, where the feed phosphorus is likely to exceed by far the requirements, and the amount of calcium is likely to be very low. In such cases an addition of calcium to the ration in the form of any of its salts except the phosphate is indicated.

The opposite condition, namely, a phosphorus deficiency, is likely to be encountered on ranges where the vegetation is low in phosphorus. In this case an addition of phosphorus to the feed is needed. This may be supplied in any convenient form, either as a feed high in phosphorus or as a mineral supplement. On the range, bone meal is the most convenient mineral supplement for this purpose. It is readily taken by cattle either alone or mixed with common table or stock salt. Since on ranges deficient in phosphorus the calcium is usually also rather low, the calcium in the bone meal is no detriment to the mineral balance. A more complicated mineral mixture containing additional mineral elements, such as the mineral-mixture salesman attempts to make the unsuspecting stock owner believe he needs, is not indicated.

References

[1]Forbes, E. B., Halverson, J. O., and Morgan, L. E.: The mineral metabolism of the milch cow. Ohio Agr. Exp. Sta. Bul. 330 (1918).

[2]Forbes, E. B., Hunt, C. H., Schulz, J. A., Winter, A. R., and Remler, R. F.: The mineral metabolism of the milch cow. Ohio Agr. Exp. Sta. Bul. 363 (1922).

[3]Miller, H. G., Yates, W. W., Jones, R. C., and Brandt, P. M.: Mineral metabolism studies with dairy cattle. Mineral equilibrium after prolonged lactation. Amer. Jour. Physiol., lxxii (1925), pp. 647-654.

[4]Fingerling, G.: Einfl. kalk-u. phosphorsäurearmer Nahrung auf die Milchsekretion. Landw. Versuchsstat., lxxv (1911), p. 1.

[5]Hart, E. B., Steenbock, H., Hoppert, C. A., and Humphrey, G. C.: Dietary factors influencing calcium assimilation. IV. The comparative efficiency of mixed green grasses and this same mixture plus steamed bone meal in maintaining calcium and

phosphorus equilibrium in milking cows. Jour. Biol. Chem., lviii (1923), p. 43.

[6]Hart, E. B., Steenbock, H., Tent, E. C., and Humphrey, G. C.: Dietary factors influencing calcium assimilation. XII. A study of the influence of hays curing with varying exposure to sunlight on the calcium metabolism of milking cows. Jour. Biol. Chem., lxxxiv (1929), p. 367.

[7]Meigs, E. B., Turner, W. A., Harding, T. S., Hartman, A. M., and Grant, F. M.: Calcium and phosphorus metabolism in dairy cows. Jour. Agr. Res., xxxii (1926), p. 833.

[8]Hart, E. B., Steenbock, H., Kletzien, S. W., and Scott, H.: Dietary factors influencing calcium assimilation. IX. Further observations on the influence of cod liver oil on calcium assimilation in lactating animals. Jour. Biol. Chem., lxxi (1926), p. 271.

[9]Hart, E. B., Steenbock, H., Elvehjem, C. A., and Scott, H.: Dietary factors influencing calcium assimilation. VII. The influence of sunlight upon calcium equilibrium in milking cows. Jour. Biol. Chem., lxvii (1926), p. 371.

[10]Hart, E. B., Steenbock, H., Scott, H., and Humphrey, G. C.: Dietary factors influencing calcium assimilation. X. The influence of ultra-violet light upon calcium and phosphorus metabolism of milking cows. Jour. Biol. Chem., lxxiii (1927), p. 59.

[11]Hart, E. B., Steenbock, H., Kline, O. L., and Humphrey, G. C.: Dietary factors influencing calcium assimilation. XIII. Influence of irradiated yeast in the calcium and phosphorus metabolism of milking cows. Jour. Biol. Chem., lxxxvi (1930), p. 145.

[12]Hess, A. F., Light, R. F., Frey, C. N., and Gross, J.: A study of the milk, blood and excreta of cows fed moderate and excessive amounts of irradiated yeast or ergosterol. Jour. Biol. Chem., xcvii (1932), p. 369.

[13]Huffman, C. F., Robinson, C. S., and Winter, O. B.: The calcium and phosphorus metabolism in heavily milking cows. Jour. Dairy Sci., xiii (1930), p. 342.

[14]Ellenberger, H. B., Newlander, J. A., and Jones, C. H.: Calcium and phosphorus requirements of dairy cows. I. Weekly balances through lactation and gestation periods. Vermont Agr. Exp. Sta. Bul. 331 (1931).

[15]Ellenberger, H. B., Newlander, J. A., and Jones, C. H.: Calcium and phosphorus requirements of dairy cows. II. Weekly balances through lactation and gestation periods. Vermont Agr. Exp. Sta. Bul. 342 (1932).

[16]Kellner, O.: Die Ernährung der landwirtschaftlichen Nutztiere. (Paul Parey, Berlin, 1924.)

[17]Forbes, E. B., French, R. B., and Letonoff, T. W.: The mineral metabolism of the beef steer. Jour. Nutri., i (1929), pp. 201-208.

[18]Theiler, A., Green, H. H., and Du Toit, P. J.: Minimum mineral requirements in cattle. Jour. Agr. Sci., xvii (1927), pp. 291-314.

[19]Theiler, A.: Untersuchungen über den Bau normaler und durch calcium- und phosphorarme Nahrung veränderter Rinderknochen. Denkschriften der Schweizerischen Naturforschenden Gesellschaft (Memoires de la Société Helvétique des Sciences Naturelles), lxviii (1932).

[20]Theiler, A., Du Toit, P. J., and Malan, A. I.: The influence of variations in the dietary phosphorus and in the calcium phosphorus ratio on the production of rickets in cattle. Onderstepoört Jour., viii (1937), pp. 375-414.

[21]Otto, J. S.: The assimilation of calcium and phosphorus by the growing bovine. Onderstepoört Jour., x (1938), pp. 281-364.

[22]Huffman, C. F., Duncan, C. W., Robinson, C. S., and Lamb, L. W.: Phosphorus requirements of dairy cattle when alfalfa furnishes the principal source of protein. Michigan Agr. Exp. Sta. Tech. Bul. 134 (1938).

[23]Archibald, J. G., and Bennett, E.: The phosphorus requirements of dairy heifers. Jour. Agr. Res., li (1935), pp. 83-96.

[24]Henderson, H. O., and Weakley, Jr., C. E.: The effect of feeding different amounts of calcium and phosphorus upon the growth and development of dairy animals. West Virginia Agr. Exp. Sta. Bul. 231 (1930).

[25]Shohl and Wolbach: Rickets in rats. XV. The effect of low-calcium high-phosphorus diets at various levels and ratios upon the production of rickets and tetany. Jour. Nutri., ii (1936), pp. 275-291.

[26]Bechdel, S. I., Landsburg, K. G., and Hill, O. J.: Rickets in calves. Pennsylvania Agr. Exp. Sta. Tech. Bul. 291 (1933).

[27]Bechdel, S. I., Hilston, N. W., Guerrant, N. B., and Dutcher, R. A.: The vitamin D requirements of dairy calves. Pennsylvania Agr. Exp. Sta. Bul. 364 (1938).

[28]Rupel, I. W., Bohstedt, G., and Hart, E. B.: Vitamin D in the nutrition of the dairy calf. Wisconsin Agr. Exp. Sta. Res. Bul. 115 (1933).

[29]Bechtel, H. E., Hallman, E. T., Huffman, C. F., and Duncan, C. W.: Pathology of rickets in dairy calves. Agr. Exp. Sta., Michigan State Coll., Tech. Bul. 150 (1936).

[30]Klimmer, M., and Schmidt, J.: Beitrag zur Ätiologie der halisteresis ossium nebst therapeutischen Bermerkungen. Monatsch. prakt. Tier., xvii (1906), p. 481.

[31]Marston, H. R.: Studies on the phosphorus requirements of sheep. Counc. Sci. & Ind. Res., Commonwealth of Australia, Bul. 85 (1934).

[32]Theiler, A.: Das Phosphordefizit und die Unfruchtbarkeit des Rindes. Schweiz. Arch. f. Tier., lxxiii (1933), pp. 47-66.

[33]Schmidt, H.: Field and laboratory notes on a fatal disease of cattle occurring on the coastal plains of Texas. Texas Agr. Exp. Sta. Bul. 319 (1924).

[34]Schmidt, H.: Feeding bone meal to range cattle on the coastal plains of Texas. Texas Agr. Exp. Sta. Bul. 344 (1926).

[35]Fraps, G. S.: Feeding values of certain feeding stuffs. Texas Agr. Exp. Sta. Bul. 245 (1919).

[36]Guilbert, H. R., and Goss: Some effects of restricted protein intake on the estrous cycle and gestation in the rat. Jour. Nutri., v (1932), pp. 251-265.

[37]Eckles, C. H., Palmer, L. S., Gullickson, T. W., Fitch, C. P., Boyd, W. L., Bishop, Lucille, and Nelson, J. W.: Effects of uncomplicated phosphorus deficiency on oestrous cycle, reproduction, and composition of tissues of mature cows. Cornell Vet., xxv (1935), pp. 22-43.

[38]Theiler, A.: Untersuchungen über den Bau normaler und durch kalzium- und phosphorarme Nahrung veränderter Rinderknochen. Schweiz. Arch. f. Tier., lxxxv (1933), pp. 339-366.

[39]Fitch, C. P., Boyd, W. L., Eckles, C. H., Gullickson, T. W., Palmer, L. S., and Kennedy, Cornelia: Report of an experiment to determine the effect of a low calcium ration on reproduction in cattle. Cornell Vet., xxii (1932), pp. 156-172.

[40]Palmer, L. S., Fitch, C. P., Gullickson, T. W., and Boyd, W. L.: Supplementary report of an experiment to determine the effect of a low calcium ration on reproduction in cattle. Effects of further reduction in calcium and of removing vitamin supplements. Cornell Vet., xxv (1935), pp. 229-246.

[41]Hart, E. B., Steenbock, H., and Humphrey, G. C.: Influence of rations restricted to the oat plant

on reproduction in cattle. Wisconsin Agr. Exp. Sta. Res. Bul. 49 (1920).

[42]Theiler, A., Green, H. H., and Du Toit, P. J.: Phosphorus in the live stock industry. Jour. Dept. Agr., Union of South Africa, viii (1924), pp. 400-504.

[43]Kleiber, M., Goss, H., and Guilbert, H. R.: Phosphorus deficiency metabolism and food utilization in beef heifers. Jour. Nutri., xii (1936), pp. 121-153.

[44]Riddell, W. H., Hughes, J. S., and Fitch, J. B.: The influence of phosphorus deficiency in dairy cows on the coefficient of digestibility and the balance of calcium and phosphorus. Jour. Agr. Res., xlviii (1934), pp. 167-170.

[45]Becker, R. B., Neal, W. W., and Shealy, A. L.: The effect of calcium deficient roughages upon milk production and welfare of dairy cows. Florida Agr. Exp. Sta. Bul. 262 (1933).

[46]Wellmann, O.: Fütterungsversuche über die Bedeutung des Mineralstoffwechsels, insbesondere der Erdalkalien und Phosphorsäure. Biologia Generalis, viii (1932), pp. 387-396.

[47]Eckles, C. H., and Gullickson, T. W.: Phosphorus deficiency in the rations of cattle. Minnesota Agr. Exp. Sta. Tech. Bul. 91 (1932).

[48]Marek, J., Wellmann, O., and Urbanyi, L.: Weitere Untersuchungen über Rachitis und Osteomalacia. Arch. f. Wiss. u. prakt. Tier., lxxiii (1938), pp. 73-82.

[49]Marek, J., and Wellmann, O.: Die Rachitis. Pathologischer Teil. (Gustav Fischer, Jena, 1931.)

[50]Marek, J., Wellmann, O., and Urbanyi, L.: Rhachitisversuche beim Kalbe und die Rachitis-Ätiologie. Arch. f. Wiss. u. prakt. Tier., lxix (1935), pp. 151-166.

[51]Lane, A. H.: Bone disease amongst horses in South Africa. Vet. Jour., n. s. 13 (1906), pp. 232-245.

[52]Theiler, A.: Die Osteoporosis des Pferdegeschlechtes. Monatsch. prakt. Tier., xvi (1908), p. 193.

[53]Ingle, H.: Osteoporosis in animals. Jour. Comp. Path. & Therap., xx (1907), p. 35.

[54]Scheunert, A., and Schattke, A.: Die Kalkarmut der Futtermittel und ihre Beziehung zur Osteomalacie der Truppenpferde. Zeit. Veterinärk., xxiii (1911), pp. 495-503.

[55]Joest, E., and Zumpke, A.: Beitrag zur Kenntnis der ostitis fibrosa des Pferdes nebst einigen Bemerkungen über die ostitis fibrosa im vergleich zu anderen Knochenerkrankungen. Zeit. f. Infekt., xxvii (1924), pp. 81-121.

[56]Niimi, K.: Experimental studies on osteomalacia in the horse. I. Etiological significance of calcium deficiency in the diet. Jour. Japanese Soc. Vet. Sci., vi (1927), pp. 273-283.

[57]Niimi, K., and Aoki, M.: Experimental studies on osteomalacia in the horse. II. Etiological significance of calcium deficiency in the diet. Trop. Vet. Bul., xvi (1928), p. 70.

[58]Kintner, J. H., and Holt, R. L.: Equine osteomalacia. Philippine Jour. Sci., xlix (1932), p. 1.

[59]Sturgess, G. W., and Crawford: Osteitis fibrosa: (Osteoporosis)—with especial reference to mineral deficiencies in the food. Administration report of the government veterinary surgeon, Ceylon (1927).

[60]Groenewald, J. W.: Osteofibrosis in equines. Onderstepoört Jour., ix (1937), pp. 601-620.

[61]Scheunert, A.: Über Knochenweiche bei Pferden und "Dysbiose der Darmflora." Zeit. f. Infekt., xxi (1920), pp. 105-121.

[62]Scheunert, A., and Hopffe, A.: Studien über ostitis fibrosa bei Pferden. II. Über die Darmflora der erkrankten Pferde und ihre Bezeihung zu den Stallvtrhältenissen. Zeit. f. Infekt., xxiii (1922), pp. 169-190.

[63]Scheunert, A., Grimmer, W., and Hopffe, A.: Studien über ostitis fibrosa bei Pferden. III. Weitere Untersuchungen über die Beziehungen von Darmflora und Darmgärungen zur Entstehung von ostitis fibrosa. Zeit. f. Infekt., xxiv (1923), pp. 85-104.

[64]Niimi, K., and Kato, K.: Experimental studies on osteomalacia in the horse. III. Effect of the treatment by means of compensation of calcium carbonate. Jour. Japanese Soc. Vet. Sci., vii (1928), p. 181.

[65]Marek, J., Wellmann, O., and Urbanyi, L.: Epiphysenlösung im Femurkopf bei jungen Pferden auf rachitischer Grundlage. Deut. Tierärztl. Wochnschr., xlvii (1939), pp. 353-356.

[66]Scheunert, A., Schattke, A., and Weise, M.: Über die Wirkung alleiniger Haferfütterung auf den Kalk- und Phosphorsäurestoffwechsel des Pferdes. Biochem. Zeit., cxxxix (1923), p. 10.

[67]Mitchell, H. H., and McClure, F. J.: Mineral nutrition of farm animals. Bul. 99, Nat'l Res. Counc. (1937).

[68]Klimmer, M.: Fütterungslehre der landwirtschaftlichen Nutztiere. Paul Parey. Berlin, 1924)

[69]Theiler, A.: The osteodystrophic diseases of domesticated animals. Vet. Jour., xc (1934), pp. 143-175.

[70]Hellmich, K.: Die Epiphysenlösung am Femurkopf bei jungen Pferden. Tierarztl. Rdsch., xliv (1938), pp. 553-540.

[71]McCrudden, F. H.: The composition of bone in osteomalacia. Amer. Jour. Physiol., xvii (1906-07), p. 32.

[72]Marek, J.: Wesen gegenseitige Beziehung und Therapie der Rachitis, der Osteomalacie und der Osteoporose. Arch. f. Wiss. u. prakt. Tier., li (1924), pp. 1-14.

[73]Lintzel, W.: Der Mineralstoffwechsel. Handbuch der Ernährung und des Stoffwechsels der landwirtschaftlichen Nutztiere, Band III (Julius Springer, Berlin, 1931).

[74]Lindsey, J. B., Archibald, J. G., and Nelson, P. R.: The calcium requirements of dairy heifers. Jour. Agr. Res., xlii (1931), pp. 883-896.

Pre-Lister Asepsis

Asepsis was never defined more succinctly than by an old Yorkshire veterinary surgeon. This man was the most successful veterinary surgeon in the north of England. Before he performed an operation, he insisted on being left alone in the farm kitchen for half an hour. What he did in that time no one knew. When at last he was on his death-bed, his son begged him to tell the secret of this success. The old man looked around the room to make sure that no one else was in earshot, then leaned forward and whispered to his son, "I boils me tools."—*From "The Story of Surgery," by Harvey Graham.*

There is no difference in the amount of energy produced by the starch contained in food and the amount produced by simple sugars. The starch, however, acts more slowly and lasts longer. The energy from a pure sugar is quickly expended.

Tuberculous Synovitis in Bovines Resulting from the Intravenous Injection of Avian Tubercle Bacilli*

By A. B. CRAWFORD, D.V.M., and A. H. FRANK, D.V.M.
Beltsville, Md.

TEN CULTURES of avian tubercle bacilli recovered from swine were received in 1936 from William H. Feldman of the Mayo Foundation, Rochester, Minn. Typing tests at our station also showed these strains to be pure cultures of avian tubercle bacilli.

In November 1936, ten yearling bulls were injected intravenously with these strains, each animal with an individual strain. The dose was 5 mg. of culture, moist weight, suspended in 5 cc. of saline solution. Six of these ten animals died of tuberculous pneumonia 31 to 54 days following inoculation. No abnormal condition was noted in the remaining four animals until July 1937, or eight months after inoculation, when swellings were observed in various joints of the extremities of two of these bulls. These enlargements, confined chiefly to the knee and hock joints, were fluctuant on palpation and appeared to be caused by a greatly increased amount of synovial fluid within the joint capsule. A clear, straw-colored fluid, with occasional flakes, was aspirated aseptically from the affected joints and from this fluid the avian tubercle bacillus was cultured. The condition of the affected animals was fair at this time.

In November 1937, one year after inoculation, it was apparent that both affected animals were losing weight. The swellings at this time were slightly larger than when first observed and on palpation were no longer doughy, but were becoming indurated. The animals walked with difficulty and reluctance and the affected joints had the appearance of becoming ankylosed. The condition of these two animals became gradually worse but, in spite of their emaciated condition, it was not until April 1939 that one of them (No. 2040) died.

The postmortem record of bull 2040 is as follows: Bull 2040 died April 23, 1939; greatly emaciated; left knee joint, left scapulohumeral joint, and right and left hock joints greatly enlarged and indurated; a vertical section of the enlargements from front to rear showed principally a thickening of the joint capsule and inflammation of the synovial membrane; in some joints the synovial fluid was clear, in others scant and discolored due to broken-down tissue and leucocytes. There was no apparent bone destruction or ankylosis, but an occasional roughened or pitted area was noted on the articular periosteum. A yellowish-brown encapsulation, 8 mm. in diameter, slightly caseous, was observed on the dorsal anterior surface of the sternum, slightly

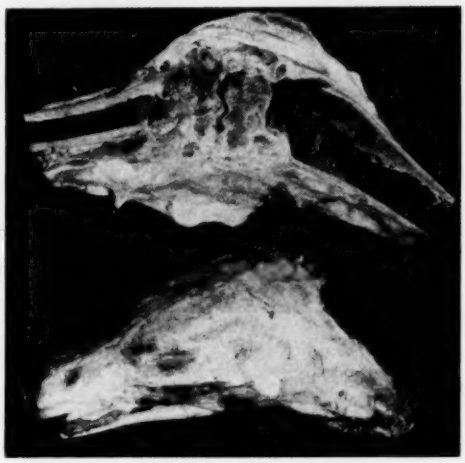
Bull 2040, showing enlargement of left knee one year after intravenous injection of avian tubercle bacilli.

*From the Animal Disease Station, Bureau of Animal Industry, U. S. Department of Agriculture.

embedded in the cartilage. The bone marrow of the left radius was slightly hemorrhagic. All organs and lymph nodes appeared normal with the exception of the internal and external iliac lymph nodes, which were one-third to one-half enlarged

Bull 2041, showing enlargements in various joints of extremities about three years after intravenous injection of avian tubercle bacilli.

and hyperplastic. Avian tubercle bacilli were recovered from the following lymph nodes: A retropharyngeal, a mesenteric, the internal and external iliacs, and the left prescapular. They also were recovered



Sagittal section of left knee joint, bull 2041.

from the bone marrow of the left radius and from the lesion on the sternum.

The remaining infected bull (No. 2041) was killed because of his poor condition on October 19, 1939. Both knee and hock joints were enlarged and indurated and had the appearance of being ankylosed. On postmortem examination all organs and lymph nodes appeared normal and the only lesions observed were in the affected joints. The general appearance of these joints on section was quite similar to that reported for animal 2040. An acid-fast organism having the cultural characteristics of avian tubercle bacilli was recovered from an affected joint.

The two remaining animals showed no abnormalities and appeared to be in good condition throughout the course of the experiment. Both were sacrificed in October 1939. On postmortem examination, one bull showed no abnormality. In the other animal, which had been castrated about a year previously by pinching the spermatic cord, the seminal vesicles were atrophied and the seminal fluid was thin, cloudy, and yellowish. Acid-fast organisms, similar culturally to avian tubercle bacilli, were cultured from this fluid. An abscess, well encapsulated, about 3 cm. (1.18 in.) in diameter, was found in the omental fat below the rectum. An acid-fast organism was recovered from this abscess, having the cultural characteristics of avian tubercle bacilli.

SUMMARY

Ten yearling bovines were inoculated intravenously with strains of avian tubercle bacilli recovered from swine. Six of the bovines died of tuberculous pneumonia within two months. Of the remaining four animals, two developed swellings in various joints of the extremities about eight months after inoculation. Avian tubercle bacilli were recovered from the synovial fluid in the affected joints. These two affected animals became progressively emaciated, one dying two and one-half years after inoculation and the other being killed in a weakened condition about three years after inoculation. The joint enlargements were found to be due to a thickening of the synovial membrane and capsule, there being no evidence of ankylosis or bone destruction. There was no evidence of organic tuberculosis in these two animals.

So-Called Shipping Fever in Dairy Cattle*

By W. C. GLENNEY, D.V.M.
Elgin, Ill.

THE SUBJECT matter of this discussion is derived principally from field experience and is based upon results obtained in a large number of cases. In some instances the writer's findings align with established facts published in authoritative journals; in other instances the findings are variable.

Hutyra, Marek and Manninger describe shipping fever as "usually an acute, more rarely subacute, infectious disease manifested by febrile disturbances and symptoms of gastroenteritis and inflammatory edema of the skin; or by inflammatory edema of the intra-alveolar tissue and necrotic croupous pneumonia with *Pasteurella boviseptica* as the causative bacterium." In the writer's locality (northeastern Illinois) edema of the skin is negligible. Pulmonary and gastrointestinal complications are the predominating diseases. Pneumonia causes the greatest number of deaths; gastroenteritis is accountable for a few.

Hemorrhagic septicemia, or so-called shipping fever, is a serious problem in the dairy-cattle industry—probably the most difficult condition encountered from September to April. It is not uncommon to see this disease enter a herd during the winter months, when the production is naturally low, and kill 5 to 15 per cent of the heavy producers.

INCIDENCE

The disease may appear any time but it causes the heaviest losses during the colder months, September to April. Although cattle of any age may be affected, this discussion will be confined to heavy pregnant and recently freshened cows.

ETIOLOGY

P. boviseptica has long been considered the causative agent of hemorrhagic septicemia. However, as this report deals with "shipping fever" in all its forms in this locality, other complicating bacteria will be enumerated.

In those spontaneous cases involving a few animals or the entire herd where no external infection has been encountered, diphtheroid streptococci seem to be the heavy offender. These cases have not been in transit in any form and without apparent reason develop a sudden rise in temperature, cough, anorexia and resulting cessation of milk flow with an occasional abortion. Diphtheroid streptococci and *B. coli* embrace the bacterial flora found in these cases. In my experience, and from laboratory reports. *P. boviseptica* has been found but rarely in these spontaneous cases, unless they are of long standing. However, in those cases resulting from exposure, change of feed and handling through transit, *P. boviseptica* is encountered in large numbers. The above-mentioned bacterial flora also may be found after the animals' resistance has been lowered to a degree where the body defenses are weakened.

PATHOGENESIS

It is difficult to explain the appearance of *P. boviseptica*, diphtheroid streptococci and *B. coli*. Careful study of field cases, laboratory reports and theories of research staffs seem to regard these causative bacteria as normal bacterial flora in a large percentage of cases. When the resistance of the animal has been lowered through exposure, change of diet, unbalanced diet or inhumane handling, this normal flora becomes pathogenic and immediately calls the body defenses into action, thus producing a temperature.

Only veterinarians actively engaged in treating these conditions from year to year can appreciate what the dairy practitioner has to contend with during the winter months. Our most difficult cases appear

*Presented at the 58th annual meeting of the Illinois State Veterinary Medical Association, Springfield, Ill., February 15-16, 1940.

when high-producing cows, heavy in pregnancy and some just calved, are trucked or shipped from neighboring states. Often a cow delivers enroute in a tightly packed truck at a temperature near zero, or she may ride hundreds of miles immediately following calving with a retained placenta and milk fever, fatal pneumonia following in a few days. The cows are moved at a crucial time and can not be handled otherwise, as the dealers buy only cows that are due to deliver soon or have just delivered. The buyers at the terminal point want only cows that are producing heavily, thereby forcing the dealers to buy as they do. This gives no time for immunization preceding shipment.

SYMPTOMS

As in any septicemia, the initial symptom is a rise in temperature. This will vary from 103° to 107° F. A few cases I have on record showed 108°. On an accurate check on 36 cases the temperature showed the incubation period to be between five and seven days, on the average. This will vary with individual resistance or immunity, the degree of virulence of infection, and the amount and severity of exposure to which the animal is subjected.

As the temperature rises there is a natural cessation of rumination and peristalsis and a diminished milk flow in lactating animals. Occasionally, a premature birth will be found but, usually, the dairy cow does exceedingly well to hold the unborn calves as nobly as she does.

Lack of regurgitation is an early symptom. Occasional too-frequent coughing is noticed. More coughing is the rule in susceptible herds where infected cows have entered. The appetite is lessened, depending more upon the length of time of sickness than upon the temperature. However, if the cases are handled successfully, they may run an intermittent temperature of 103° to 106°, and every animal will eat surprisingly well. In one particular herd that was treated for a total of twelve days before the temperatures were normal, there was not a single animal that missed a meal, even though the temperatures were fluctuating heavily at times.

Untreated cases that terminate fatally will develop rapid breathing on the third to the sixth day. As the lung complications increase, the animal will stand or lie with extended head and breathe through the mouth. A respiratory grunt or groan is common in an infected herd with advanced necrotic croupous pneumonia. The front and rear legs may "buckle," giving the animal the appearance of contracted tendons. When the symptoms just mentioned are encountered, do not attempt treatment, as it will be of no avail. Death will come in 24 to 48 hours following a loss of appetite, protracted illness and temperature and respiratory complications.

Occasionally, the temperature will break early preceding pulmonary complications and a severe diarrhea will develop. This is the exception rather than the rule in my experience and usually means a long, drawn out case that may live or die, and will be as well off either way. The animal becomes badly emaciated and even though the treatment be complimentary, it often will not regain normal health.

In the literature an edematous form is described but I have not encountered this condition and will not attempt to discuss it.

PROGNOSIS

The prognosis in each case varies with the length of time the animal has been sick, the degree of pulmonary complications, the coöperation of the owner, the accessibility to treatment and, to a certain extent, the value of the animal.

The literature gives a high mortality—75 to 95 per cent. I have no doubt that in days gone by the mortality was that high. However, with the advancement of medicine, this has been lowered significantly. When called early the practitioner can save 80 to 90 per cent of the cases. If he is called a few days after the animal has been affected, there will probably be 75 to 85 per cent recoveries. But, when the patient refuses food, is breathing rapidly and heavily with extended head, the mortality will be 100 per cent.

Often a practitioner will be misled by the temperature. It may read normal whereas in reality it is going from 106° to 55°, which is the barn temperature. The general condition of the animal should always be considered along with the temperature.

In my locality one is often praised as much for prognosing as for diagnosing and treating. With a poor prognosis the animals are immediately shipped, if they are in a condition to ride to market. With a fair to good prognosis, treatment is instituted until improvement is noted and, if there is no response, the animals are shipped. Whether or not it is good ethics to allow shipment on a poor prognosis is debatable, but common sense tells one that infected animals should not be slaughtered. However, regardless of what is advised, the cattle will be salvaged for what they will bring, as the owner knows he has no other recourse in order to save his investment.

Diagnosis

Do not depend on a single sick animal in a herd of 35 to 40 for a diagnosis. Take the temperature of every animal in the herd. One high temperature, unless the animal has recently been shipped or trucked, does not indicate shipping fever. However, if 10 to 20 per cent of the herd is registering 103° and over, and there is a history of recent movement of animals, institute treatment at once. Climbing temperature and coughing are usually present. The history that new cows have entered the herd, even though they appear normal, may signify that immune carriers are present. This is partially diagnostic. In the early stages the appetite will usually be fair to good and is not diagnostic. As the disease progresses and lung complications develop, there is no mistake in diagnosis.

The spontaneous cases with no outside exposure develop in a similar manner—rise in temperature, coughing, cessation of milk flow—but usually show a marked diarrhea at the beginning. This is usually caused by a chilled barn, or the animals have been chilled outside the barn while the stables were being cleaned. It responds more rapidly to treatment than true hemorrhagic septicemia.

Once a practitioner has encountered this disease, he will have no trouble in dealing with a similar condition at a later date.

Nasal and lacrimal discharge of a serous nature and later turning to a purulent consistency is common. Shivering and cessation of regurgitation are noticed. General listlessness prevails and the mucous membranes show a generalized hyperemia.

If in doubt on a diagnosis, procure laboratory determinations as early as possible by sending in liver, lung and spleen specimens packed in borax powder. The state or commercial laboratory will coöperate in this. Such laboratory determinations will aid in specific treatment, which is essential for a favorable outcome.

Spontaneous cases will reveal a predominance of streptococci, and for this condition my clients are familar with a diagnosis of acute streptococcic pneumonia. Cases resulting strictly from shipment and which seem to be of a contagious nature in a susceptible herd are diagnosed as hemorrhagic septicemia, or the common layety term "shipping fever."

When dealing with the ordinary dealer and buyer, the practitioner should be cautious on diagnosis. A shipping-fever diagnosis incriminates the dealer, and the buyer often will stop at no end to collect full damages. This may amount to hundreds or thousands of dollars. A diplomatic practitioner will save a great many legal disputes by carefully handling both parties and effecting a suitable compromise. It is his duty to the client and to the profession to handle such situations as tactfully as possible.

Prophylaxis

As previously mentioned, establishing an immunity previous to exposure is impossible because the dealers usually buy and ship immediately. Vaccination at the time of shipment is of little value and at times seems to accentuate the disease. Hemorrhagic septicemia serum is the biological product of choice preceding shipment, but

the use of it has been discouraging, not to mention the cost.

Pulmonary mixed bacterins theoretically should be of value in prophylaxis. In my practice they are the product of choice if a week or two can be allowed for immunization, but of no value for immediate protection. However, as most animals are given heavy repeated mixed bacterin doses following exposure, it is wise to give the initial dose of 5 to 10 cc. at the time of shipment and to follow this twice a week with an increased dosage, until recovery or immunization is apparent.

Soda has been used in large amounts prior to shipment, with little success. It does seem to have a slight value for treatment.

TREATMENT

Due to the indefinite results from biological products, the choice here should be made through a fair consideration of all factors involved. There is no specific biological treatment; hence, the product selected should have a definite ratio between results and cost. This rules out everything but pulmonary mixed bacterin. It is inexpensive and produces results as good as vaccines, aggressins and serums. Repeated injections twice a week as long as treatment is instituted are beneficial. My practice is to inject all animals in the barn twice weekly until new cases have ceased. The initial injection consists of 5 cc. subcutaneously. This employs a test dose for any animals that are allergic to this product. The second injection consists of 10 cc. and, thereafter, up to 40 cc. at a single injection, depending upon the individual animal. Usually results will be forthcoming after two injections, if at all. Two or three injections three to four days apart will immunize the susceptible animals in the barn.

Biological treatment should be supplemented by specific pharmaceuticals. The "all-American c u r e - a l l," sulfanilamide, seems to be the most popular of these at present. I have used it alone, with soda and with biological products. After finding a high percentage of streptococci in laboratory examinations, I have used it with variable but encouraging results. It is not the so-called cure-all, but it certainly warrants a trial.

The use of sulfanilamide alone in spontaneous cases within a herd where the history reveals no movement of animals has given me fine results. Fifteen and five-tenths to 17.8 Gm. (240 to 275 gr.) three times daily in capsule or bolus usually quiets down a chilled herd in two or three days. The use of similar doses of soda seems to be only slightly beneficial. To satisfy the owner, 5 to 10 cc. of pulmonary mixed bacterin is given the first day of treatment. When the temperatures are normal and appetites are lacking, use a good tonic or pulmonary specific in capsule or drench to complete the treatment. These cases, as a rule, do not present any great difficulty.

The test of the practitioner comes on the true shipping-fever cases. I have recorded 36 typical cases with pulmonary mixed bacterin, sulfanilamide and soda treatment. These animals received 5 to 10 cc. of bacterin preceding shipment of several hundred miles. Five to seven days later all showed temperatures of 102 to 106°. Sulfanilamide was used in daily doses of 48.6 to 97.5 Gm. (750 to 1,500 gr.). The average dose was three 30-Gm. (1-oz.) capsules of sulfanilamide daily, with a similar amount of soda. When the temperature was 102° or below, treatment was discontinued. Often a case relapsed and further treatment was necessary. Of these 36 cases, the average time of treatment was six days. The average amount of sulfanilamide given was 180 Gm. (2,760 gr.), and the same of soda, at an average cost of $1.15 per cow. Three of the 36 animals failed to respond to any treatment. This gives approximately 10 per cent mortality as compared with the suggested 85 to 95 per cent mortality. From these results it appears that this treatment is the best at hand.

An occasional case that did not respond to the 48.6- to 58.5-Gm. (750- to 900-gr.) dose daily received as high as 97.5 Gm. (1,500 gr.) daily without any ill effect. The

results of soda do not warrant any great credit.

Converse, dealing with sulfanilamide treatment in humans, warned against the insufficient time of treatment. His observations show that sulfanilamide reduces the virulence of the hemolytic streptococci, not necessarily destroying the bacteria, and with early discontinuance of the drug, the virulence may return. Streptococcic antibodies are not developed in the animal body with the use of sulfanilamide. Therefore, in using this drug, a maximum dosage and maximum time limit are necessary to avoid subsequent temperature rise and reinfection manifestations. Notwithstanding the necessity of ample dosage, all animals should be kept under close observation to detect toxic symptoms that may arise. Close observation for incompatibilities, such as magnesium sulfate, is essential.

Nux vomica was used along with a pulmonary specific to good effect on slow cases that carried a normal temperature. Autogenous bacterins and pulmonary diathermy may be of benefit, but I have not tried them. Mustard plasters have proved futile. Spirits of fermenti at times give good supportive treatment.

CONCLUSIONS

Shipping fever in dairy cows is the dealers' and practitioners' worst enemy during the winter months, particularly the pulmonary form.

Only specific treatment produces results. Bacterial examinations are necessary to institute this.

Biological products are of questionable value unless an early period of immunization is established preceding shipment. Even then, the results are discouraging.

Pulmonary mixed bacterins and sulfanilamide give the most promising results in treatment. Sulfanilamide is not a "cure-all" but it warrants further use in bovine pulmonary disorders in which streptococci predominate.

Autogenous bacterins and pulmonary diathermy also warrant further trial.

Color Photomicrography

Extremely instructive to those interested in the preparation of colored photomicrographs is an article entitled "A Technique for Making Photomicrographic Prints in Color" which appeared in *The Journal of Technical Methods and Bulletin of the Internal Association of Medical Museums*, No. xix, 1939. The author is Hilton A. Smith, D.V.M., Division of Veterinary Science, Texas A. & M. College, College Station, Texas.

Elephants

Elephants are susceptible to foot-and-mouth disease. Vesicles affect the mucosa of the trunk, the tongue, the mouth and the toes. Other diseases of elephants common in other domestic animals are anthrax and a variola of unknown relationship.

The elephant's skin if 18 to 25 mm. (0.7 to 1 in.) thick and the panniculus carnosis is heavy and powerful. Suturing cutaneous wounds should be avoided unless absolutely necessary, as the sutured skin does not behave well. When suturing is done, silver wire should be used in the form of interrupted sutures set 3 to 5 cm. (1.2 to 2 in.) apart. (*Abst., The Veterinary Bulletin, x, February 1940, p. 110.*)

Frozen Sleep (= Crymotherapy)

Crymotherapy, the use of cold in therapeutics, carried to the extreme of reducing the body temperature 10 to 18° F. for several hours to two or three days, is employed in the treatment of human cancer. The suffering is relieved during the treatment and modified in severity for varying periods thereafter.

The same treatment has been employed in the treatment of drug addiction and other conditions but in no case is it yet regarded as a cure.

"These treatments," says *Science News Letter*, have been rechristened with the technical term, crymotherapy." The word, however, is not a stranger in veterinary literature. The old professors used the word for ice packs, ether sprays, and even flushing the heatstroke case with the hose.

Diseases of the Digestive System in Bovines*

By M. G. FINCHER, D.V.M.
Ithaca, N. Y.

IT IS my primary purpose to describe indigestion in cattle and to refer to other diseases with which this condition may be confused. Synonyms commonly applied to the disease that will be discussed are acute indigestion, overloading of the fore stomachs, impaction of the omasum or abomasum, acute tympany, atony of the fore stomachs, dilatation of the rumen, or acute indigestion with tympany. Thus, it is seen that indigestion used in this broad sense will include nearly all digestive disturbances.

Acute indigestion of cattle is defined in Udall's[1] text as follows:

> Acute and subacute indigestion of ruminants is an atony of the fore stomachs induced by overloading of the rumen and reticulum with food or indigestible substances; depending on the kind of material, it leads to impaction of the rumen, tympany or gastrointestinal catarrh. In rare cases an excessive amount accumulates in the omasum or abomasum.

CAUSES OF DISTURBED DIGESTION

In order to approach the subject of disturbed digestion in cattle it is essential to describe some of the causes that operate, at least in the eastern part of the United States.

Overfeeding is one of the most common causes among cattle kept in good herds where forced production is in vogue. The cattle in these herds are given all the food that they can possibly ingest. A manger full of excellent hay usually is left untouched because they have had more concentrates, silage, molasses and beet pulp than was needed. In a cow whose diet is as abundant as this any slight change in the quality of the foodstuff may result in anorexia, constipation, diarrhea and reduced production.

Cattle being prepared for the show circuit are occasionally forced beyond their capacity. In one young bull, for example, I saw repeated severe tympany and dilatation of the rumen during two consecutive summers while the bull was being forced for growth and flesh. For several years since that time the bull has remained normal when fed moderately. Strangely, in this bull no change in diet or medication would overcome these repeated attacks of bloat. Lobeline sulfate given subcutaneously twice daily in 0.1-gr. doses likewise failed to arrest the condition, but while this remedy was at hand, the bull could still be forced.

Unlimited eating of food to which they are not accustomed is most often observed in cattle owned by a poor or inexperienced farmer. Recently, a family cow that was receiving very little to eat broke loose, traveled to a neighbor's hog barn and ate several quarts of finely ground wheat middlings. The animal was dying from gastroenteritis and toxemia three days later when we were called. In the fall cattle often break loose and consume large amounts of half-ripe corn on the cob, apples, threshed grain, etc.

Indigestible and damaged food, such as chaff, frozen grass or silage, bean pods, and straw that is decomposed or mixed with manure, may in rare cases be recognized as an important cause. In a cow that stands next to the hay shaft where all the fine leaves and chaff are allowed to shake out of the hay, one should not be surprised to find a persistent indigestion. This may be associated with an impaction of the fourth stomach.

Eating indigestible substances, such as the placenta, has caused little trouble in cows that I have seen. However, when grass or hay is run through a silage cutter

*From the department of medicine, obstetrics and ambulatory clinic, New York State Veterinary College at Cornell University; presented at the 40th annual conference of veterinarians at the University of Pennsylvania, Philadelphia, Pa., January 5, 1940.

and put in the barn in the dry state, or in a silo with molasses or phosphoric acid, great care must be taken to see that all of the wire fence and baling wire are kept off the meadows from which the grass or hay to be chopped is obtained. In at least two large herds there occurred what seemed to be a great increase in the incidence of indigestion from foreign bodies after the practice of chopping hay was started. The wire is cut so finely by this method that the cows are virtually forced to swallow one-half to 3-in. lengths of wire with the chopped hay.

There are many other causes of indigestion in cattle, such as a sudden change of food or feeding improper amounts of roughage to young animals under 6 months of age. Predisposing causes are chronic peritonitis from traumatic gastritis, fatigue and advanced pregnancy.

The seasonal influence on indigestion is very clearly demonstrated by the large number of cases that the practitioner in the East is required to treat in October and November. During the winter months, when cattle receive less exercise and more dry food, indigestion is much more common than when the animals are outdoors day and night on good pasture.

Symptoms

In any discussion of this subject one should be guided by several cardinal signs that may be detected even by a well-trained attendant.

The patient will regularly show partial or complete loss of appetite and dullness. The evacuations are usually scanty, rumination has ceased and tympany may be present. In rare cases emesis is noted.

The definite history that the cow or herd has broken from the stable or pasture and has had a chance to consume some definitely injurious type of food is very important. If the history does not indicate that any of the above causes of indigestion are operating, then the animal should be more thoroughly examined for symptoms that may possibly suggest indigestion and exclude other diseases. In other words, where a cow has suppressed rumination and constipation, colic or diarrhea, it is easy, without a careful examination, to issue a diagnosis of simple indigestion. In a high percentage of cases that diagnosis will be correct. The digestive tract may be disturbed, however, accompanying many other diseases.

In trying to detect symptoms that suggest the kind of indigestion with which we are dealing, we rely on the history, pulse, temperature, and the use of a phonendoscope to detect rumen contractions, peristalsis, and heart action. We rely very much on the absence of extreme soreness on percussion with the thumb or clenched fist, or even with a hammer and pleximeter. Last of all, a vaginal or rectal examination may be of extreme value.

The importance of the pulse can not be overrated. But it must be kept in mind that nervous animals show a high pulse rate just from the excitement of having a hand placed on the lateral surface of the mandible. This is the best place to make a true estimate of the rate and character of the cow's pulse. In a few unusually wild animals the caudal artery may be used or the pulse should be counted at the jaw for a sufficiently long time to allow the nervous cow to quiet down, so that one may make a true estimate of the rate. In general, if there is a strong pulse of 60 to 70, or less, the condition is not grave and it may be simple uncomplicated indigestion. A higher pulse rate and a weak pulse suggest serious toxemia and many other complications, such as traumatic gastritis with or without pericarditis, or gastroenteritis. The pulse is usually fast and weak, however, when fermentable material or large quantities of grain have been eaten.

A temperature of 95°-101° F. usually accompanies those indigestion cases caused by overeating on apples, corn and corn fodder. It is seldom higher than 104° F. in uncomplicated indigestion. Cows that have eaten large amounts of ground feed and are in danger of dying in a few days may carry a normal temperature.

Pain caused by indigestion is usually shown in cattle by grunting or moaning, and more rarely by colic. Staggering is frequent when large amounts of apples or corn have been eaten, and many of these cows go down in a coma. They then resem-

ble a cow with milk fever or some severe poisoning.

The size of the abdomen will depend on the amount of tympany and the amount of food ingested. The more highly fatal cases may be gaunt after they have been sick for several days. In these cases there is great variation in the character of the rumen contents. The rumen may contain gas above a layer of food, or a thin, soft foamy mass of food and gas, or a hard or doughy mass of more or less impacted food in the left flank region.

Rumen contractions which may normally be felt with the hand in the left flank or heard with a phonendoscope are absent in severe indigestion or occur less frequently (1 per minute or 1 in 3 minutes) if the function of the fore stomachs is mildly disturbed.

Pain may be present on percussion of any part of the digestive tract during simple indigestion and perhaps indicates the presence of an inflammation of the part where greatest soreness is noticed. The pain induced by percussion in these cases of indigestion is seldom detected as easily as the soreness that accompanies circumscribed peritonitis with traumatic gastritis.

During several years there have come to my attention a few cases of impaction of the abomasum. In these I have been able, by pressing upward with the clenched fist, to find a great increase in the tension and weight over the floor of the abdomen posterior and slightly to the right of the xiphoid cartilage. But it is difficult to be sure that one is not lifting upon a fetus or even the overloaded omasum. I have seldom determined any evidence of pain over this area, but the abomasum seems hard to lift at this point. Persistent constipation and more or less tympany are often present for many days.

In going over the right side of the cow we look largely for pain on percussion or palpation over the liver and intestine. The absence of any peristaltic sounds in the right flank is a cause for concern, since it may mean that there is a severe enteritis. A greatly increased peristalsis is not as alarming, since it means some slight irritation of the intestinal tract or bowel catarrh.

A study of the bowel evacuations will show constipation or scanty evacuations. The presence of fetid feces with more or less mucus or blood suggests bowel catarrh. Liquid feces are regularly present in calves suffering from inflammation of the bowels in varying degree and in cows that have been turned on lush grass in the spring.

A rectal examination is seldom of very much real value in indigestion in cattle except to determine the degree of impaction and distention of the various parts of the digestive tract. It is of extreme value when one attempts to palpate an intussusception or other rare displacement of the intestine. One may also obtain samples of feces and rectal mucosa for examination for Johne's disease and coccidiosis by this method. A rectal or vaginal examination should usually be made in cows that show colic, kicking at the flank, lying down and rising. It is possible by vaginal examination to find one or both ureters enlarged, and this is done most easily by a vaginal examination. The two enlarged ureters stand out like large cords anterior to the urethra beneath the vaginal floor. The ureters are more easily detected in well-marked pyelonephritis. The colic should then be attributable to pyelonephritis due to *Corynebacterium renalis*. An examination of the urine also may be needed to verify the diagnosis. On rectal examination the enlarged left kidney and ureter may be palpated. In a routine rectal examination in a cow suspected of having indigestion one may occasionally be surprised to find that the uterus is the real source of trouble.

Metritis with septicemia, and dropsy of the fetal membranes, during pregnancy may cause a depressed digestive activity. In one instance I trocarized the left flank in a cow that was reported by the owner to be staggering from overeating on corn in the fall. Fetal fluids returned through the trocar. The cow was suffering from a fatal case of dropsy of the amnion and allantois with twin pregnancy.

COURSE AND TERMINATION

The course in indigestion varies from a highly fatal apoplectic attack in calves to

cases in cows that linger for weeks. Acute tympany accompanying alfalfa bloat or bloat from irrigated ladino clover pasture, as shown by Dougherty,[2] ends fatally in a few hours unless relieved. In cows that have overloaded on heavy-ground food or other kinds of diet, death may ensue in two to four days from hemorrhagic gastroenteritis. Overloading or impaction of the abomasum, if it occurs even rarely as a primary, uncomplicated condition, tends to terminate fatally after several weeks or longer. It is usually complicated by chronic adhesive peritonitis from a foreign body.

Most cases of indigestion in cows tend to recover, especially if given suitable, early treatment. Even cows having apple indigestion with such a severe toxemia that they stagger may recover spontaneously; but if coma develops from apples or green ears and stalks of corn in the fall and goes untreated, death often promptly follows. Indigestion in calves under 6 months of age is serious since their rumen is not able to handle roughage.

DIAGNOSIS

Usually indigestion can be readily diagnosed by the history and symptoms. It is true, however, that many cases are diagnosed as indigestion on a hurried single examination, whereas a later examination would show the case to be something least suspected.

Prompt and complete recovery after treatment is suggestive of uncomplicated indigestion. A partial recovery with a repetition of the symptoms points to a traumatic gastritis with an involvement of the peritoneum, liver, spleen or heart. A loss of condition often may mean that traumatic gastritis is present.

The symptoms that suggest diffuse peritonitis accompanying traumatic gastritis are: The cow becomes worse after advanced pregnancy and parturition; anorexia is more often complete and persistent; scant, watery or mucous evacuations are the only result of a thorough use of laxatives and rumen stimulants; tympany may persist for several days and the gas may be outside the rumen; pain is often present on kneading of any part of the abdomen and is accompanied by shallow breathing, painful expiratory grunt, recumbency, stiff, careful movements when walking, slight daily increase in the pulse, and a sudden decrease in the milk flow. These severe symptoms in animals on natural pasture should lead one to suspect traumatic gastritis with peritonitis.

Acetonemia will cause a rapid loss of condition and nearly all of the symptoms of indigestion. The digestive form of acetonemia occurs near the termination of pregnancy and during the first two months after parturition. It may be positively diagnosed by the simple Ross test. During the several years that this test has been employed, many of the cases of "chronic indigestion" and of so-called "intestinal catarrh" in cattle have received the diagnosis of acetonemia.

In addition, digestive disturbances with pronounced atony of the fore stomachs are observed in nearly all febrile conditions, such as malignant head catarrh (catarrhal fever), metritis, or mastitis and acute affections of the respiratory tract. Even post-partum hemoglobinemia, as described by Farquharson[3] and others,[4] might be mistaken for indigestion. The distinctly pale mucosae, the red urine, and a blood count should assist one in reaching a correct diagnosis in this rare disease.

TREATMENT

No particularly new treatments for indigestion have been brought to my attention. The first thing of importance when a call is received for a cow that is bloated is to advise the owner to put a gag in the mouth of the animal and, if possible, to stand the cow with the rear parts lower, or to force the patient to move until assistance arrives. (It is stated that a young calf may regurgitate gas more freely if put on its back.) These procedures will save a life now and then, whether the tympany is from indigestion or choke. Laxatives, rumen stimulants, carminatives, antiferments and general stimulants are about all that most cases require. Rarely, surgical interference is necessary.

The removal of the contents of the digestive tract by laxatives and other medi-

cation has been quite successful in the ambulatory clinic, and during the past 20 years I can recall but few cases which were treated by rumenotomy. In one instance, I removed four bushels of apples and other rumen contents from a very large, thrifty, vigorous Holstein cow. I then explored the mucosa of the reticulum and was surprised to find a wire perforating it and circumscribed peritonitis present. The cow responded well. No doubt a rumenotomy would often provide more prompt and satisfactory relief for acute indigestion, but I believe it is seldom necessary in New York State, except for the relief of selected cases of peritonitis due to a foreign body.

A satisfactory recovery will follow the removal of the weight of the rumen contents and the foreign body in over half of the cases of traumatic gastritis that are suitable for operation, as shown by Bardwell[5] and Gibbons.[6] A cow should seldom be operated for traumatic gastritis while her pulse is as high as 90 per minute. Severe cases of rather extensive acute or chronic peritonitis will recover, although much peritoneal exudate may flow from the infected incision several days after the operation. Rumenotomy as a treatment for our cases of indigestion has been largely confined to that group of cases where the digestive disturbances were secondary to traumatic gastritis. In three out of about 100 cases that have been operated in this clinic since 1926, abortion followed in 38 to 50 days. All three had traumatic gastritis with extensive peritonitis. Two of the three calved normally one year later, and at present appear to be in excellent condition.

As a laxative in indigestion we usually employ 12 Gm. (180 gr.), or less, of tartar emetic powder. This is weighed and a dose is carried in a glass vial. We dissolve this amount in several liters of warm water and pump it into the rumen through a colt stomach tube passed *via* the nostril. This may be repeated in a few hours. It is also convenient and effective to add to the water one or all of the following carminatives and antiferments: Aromatic spirits of ammonia, 1 to 3 oz.; creguiatone, respirol or creolin (Pearsons), 1 to 4 dr.; F. E. or tincture of capsicum, 1 to 4 dr.; soluble pine oil, 1 to 2 oz.; turpentine, 0.5 to 1 oz.; and, rarely, formalin, 0.5 oz.

In addition, one may give 1 to 2 lbs. of Epsom salt, but we often prefer to use 1 to 4 qts. of mineral oil. Other laxative oils, such as 1 pt. of castor oil, 1 to 2 pts. of Wesson oil, or a large amount of melted lard may be useful. Many of our cows have received 1 or 2 lbs. or more of Epsom salt a day or two before we are called. In these we prefer to employ large doses of warm mineral oil or tartar emetic in small doses plus warm water and mineral oil. In the ambulatory clinic raw linseed oil has not proved very useful as a laxative for cows, although it is frequently used by others with apparently excellent results.

A cheap and fairly effective treatment in a mild case of indigestion is 1 to 2 lbs. of Epsom salt with 1 to 5 gallons of hot water. In addition, any cow with indigestion will respond more promptly if given 0.5 to 1 gr. of strychnine sulfate by the mouth every three to six hours for a few days. The combination of an equal amount of arsenic trioxide with the strychnine in an aqueous hydrochloric acid solution, while it may not be indicated or necessary, has proved to be sufficient by itself to take care of most mild cases of indigestion.

In a few of the larger herds it is agreed with the owner or herdsman that nearly all cows that appear to have indigestion are to be given 0.5 oz. three or four times daily of the following tonic:

Arsenic trioxide	10 Gm.
Acid hydrochloric, dil.	50 cc.
Water to make	1,000 cc.
Strychnine sulfate	10 Gm.
Acid hydrochloric, dil.	50 cc.
Water to make	1,000 cc.

Mix equal parts of these two solutions, thus making each half ounce contain about .065 Gm. (1 gr.) each of strychnine and arsenic. We feel that to use this gives better results and is safer than to allow the layman to drench the cow with oil or salts.

We rarely use any rumenitories except the above-mentioned large dose of tartar emetic and an occasional .75-gr. dose of eserine salicylate, as suggested by Amadon.[7] We used barium chloride for years, along

with salts and tartar emetic. The results are just as good, or perhaps better, since we ceased to employ barium chloride. The stomach tube is used more often, thus introducing large amounts of water and removing more or less gas.

Fewer cases require trocarization if a large stomach tube is passed through the mouth, with a metal tube or Emont's speculum to protect the tube. The colt stomach tube passed by the nostril will often fail to cause the cow to regurgitate and gas will not return *via* the tube, although this is the easiest and quickest way for me to pass a tube. When it is difficult to be certain where the stomach tube has gone, have someone blow on it and listen to the air bubble into the rumen. This may then be removed and a larger tube put down through the mouth, with the result that a surprisingly large amount of gas will be expelled from the rumen. Trocarization has the advantage of giving prompt relief if the gas is not too thoroughly loaded or mixed with semi-liquid food particles, but an annoying subcutaneous emphysema often occurs at the time of trocarization.

We have been unsuccessful in using the steam hose as a means of removing rumen contents. This tube with a 1.5-in. lumen is passed through a wooden gag in the mouth. The hose may be churned in and out of the stomach until a large amount of food is pumped or lavaged out of the rumen. It may be necessary to introduce warm water through a smaller stomach tube inside the steam hose. W. F. Guard of The Ohio State University and others in personal communication have reported excellent results by this method.

One case, a 10-year-old Holstein, treated on December 25, 1939, was an ideal cow on which to have used this. She was found on a farm where little grain is given the cattle. This cow broke loose and ate 1½ bushels or more of oats and barley and a few quarts of 20 per cent protein milk maker. She was given no treatment for 31 hours but was allowed to fill up on ice water after she had staggered to a nearby creek.

Her pulse was 80 to 95 and weak. The temperature was 101.5° F. She was able to stand but weaved when turning or walking. The abdomen was large yet the rumen was not tight enough for trocarization. It was contracting very feebly. The intestine was full of gas and food and rather inactive. Few, thin feces mixed with gas were passed. The milk flow had ceased although the cow was fresh but two months and had been producing 40 lbs. daily.

A stomach tube was passed by the mouth through the tube of an Emont's speculum. Six quarts of mineral oil, 1 qt. of Wesson oil, and 5 gals. of warm water containing antiferments and carminatives were given. Much gas was returned during several minutes that the tube was left in the stomach. It was also assumed in this case, as in all cases where there is paresis, paraplegia, or coma, that calcium gluconate was indicated. The cow received 1,500 cc. (3 pts.) of a 20 per cent solution per vein (300 Gm.). One ounce of the arsenic and strychnine tonic was given by the mouth and 0.5 oz. prescribed every three to five hours. No return call was needed, but one week later I made a visit to observe the result. Huge quantities of soft feces had been passed for several days and the cow had now returned to one half her amount of milk and a fair appetite. Thirty days after she over-ate, she was almost normal. She was still given tonic and the milk flow was good.

Obviously, this cow had been sick a little too long for a rumenotomy. I did not employ the steam hose, but it would have been better in this case, as it probably would have siphoned out much of the contents of the rumen at once.

In a few cases of coma, weakness and poisoning following unlimited consumption of half-ripe corn on the cob and green corn fodder in the fall of 1939, I apparently secured results with 50 cc. (1.66 oz.) per vein of sodium thiosulfate, 30 per cent, and sodium nitrite, 2 per cent, when the usual treatment consisting of large doses of calcium gluconate per vein had failed to give the desired results. Two of ten cattle affected in one herd died promptly as though poisoned by prussic acid or something else from the corn. Usually, in indigestion from large quantities of apples or corn, we have found that 1,000 to 1,500 cc.

(2 to 3 pts.) of 20 per cent calcium gluconate overcomes the coma promptly. Sometimes .25 gr. of atrophine sulfate has been useful here. Ten years ago we obtained about the same results by udder inflation. Autopsies in this type of case show hemorrhagic gastroenteritis. In rather severe cases of enteritis we feel that there may be value in 1,000 to 4,000 cc. (1 to 4 qts.) of normal saline given per vein daily for a few days.

More important than treatment is the ability to follow up the cases which one considers to be indigestion, in order to determine the true diagnosis. Many of them will look like indigestion on the first call but will prove to be something as far removed as malignant head catarrh or pyelonephritis if followed through to a final diagnosis.

References

[1]Udall, D. H.: The Practice of Veterinary Medicine (3rd ed., D. H. Udall, Ithaca, N. Y., 1939), p. 90.

[2]Dougherty, R. W.: Physiological studies of induced and natural bloat in dairy cattle. Jour. A.V.M.A., xcvi (Jan. 1940), p. 43.

[3]Farquharson, J., and Smith, K. W.: Post-parturient hemoglobinuria of cattle. Jour. A.V.M.A., xciii (1938), n. s. 46 (1), p. 37.

[4]Madsen, D. E., and Nielsen, H. M.: Parturient hemoglobinemia of dairy cows. Jour. A.V.M.A., xciv (1939), n. s. 47 (6), p. 577.

[5]Bardwell, R. H., and Udall, D. H.: The diagnosis and treatment of traumatic gastritis. Cornell Vet., xxvii (1937), p. 302.

[6]Gibbons, W. J.: Traumatic gastritis. Cornell Vet., xxii (1932), p. 342.

[7]Amadon, R. S.: An experimental study of drugs stimulating the motility of the ruminant stomach. Jour. A.V.M.A., lxxvi (1930), n. s. 29 (1), p. 65.

Sulfanilamide is catalogued with iodine, arsenic, mercury, quinine, and other dyes; nicotinic acid with cod liver oil, calcium and other minerals, wheat germ oil, and carotene—agents which maintain the nutritive equilibrium.

A strain of western type encephalomyelitis virus was successfully propagated in the developing embryos of the chick, robin, pheasant, goose, pigeon, turkey, sparrow, duck and guinea fowl. The infected embryos of the chick, robin, pheasant and goose gave a titre of 10^{-16}; the sparrow and turkey, 10^{-12}; the duck, 10^{-9}; and the guinea fowl, 10^{-8}. (*Mitchell, Walker and McKercher. Canadian Journal of Comparative Medicine, iii, November 1939, pp. 308-309.*)

The Nature of Bang's Disease

Agglutinins appear in the blood within a period of two weeks to five months following infection. Not more than 30 per cent of cows abort after exposure to the infection. In a herd observed over a period of 15 years, 6 per cent of the reacting animals failed to conceive. Sixty-seven per cent of the reacting cattle aborted one or more times. Sixty-four per cent of the aborters aborted once and 28 per cent of these became sterile. Twenty-three per cent of the aborters aborted twice and one third of them became sterile. Five per cent aborted three times and 5 per cent aborted four times. The most susceptible period is the last half of the gestation period. Cows which become infected when not pregnant rarely abort.

Brucella abortus infection produces acute and chronic placentitis, fetal pneumonia and septicemia, endometritis, choriotis and mastitis. Sterility is an important aftermath of the infection. An examination of the fetal membranes at the termination of the pregnancy will usually give one an idea as to the condition of the uterus. The infected placentas are dull, yellowish, opaque and necrotic. Normal placentas are pinkish and glistening. Only parts of an individual placenta may show the characteristic lesions of the infection.

The fetal placentas of one cow were studied through four gestation periods. The first pregnancy showed extensive placentitis and endometritis. In the second, the placentas were normal but reduced in number by one half. The fetal placentas expelled after the termination of the third pregnancy were normal in number, and only a few were diseased. The fourth-period placentas were normal in every respect. *(Abstract of a paper presented by E. T. Hallman, D.V.M., at the annual meeting of the Missouri Veterinary Medical Association, Columbia, Mo., January 31 to February 2, 1940.)*

While fluorine in excessive amount is harmful to enamel formation, a certain quantity of it is necessary to prevent caries.

The Control of Bang's Disease in an Experimental Herd of Range Cattle*

By H. MARSH, D.V.M., and H. WELCH, D.V.M.
Bozeman, Mont.

IT SEEMS evident that the methods which have been evolved for the control of Bang's disease in dairy and purebred cattle should be modified to make them applicable to the range and semi-range conditions under which the beef cattle of the West are handled. The Montana Veterinary Research Laboratory has had the opportunity, through the coöperation of the United States Range Livestock Experiment Station at Miles City, to obtain some interesting data pertaining to the control of Bang's disease in range cattle.

The U. S. range station has maintained a herd of Hereford cows under range conditions since 1924. Bang's disease had probably existed in this herd since it was founded, but the number of abortions had been small. It was thought that an opportunity existed here to test the hypothesis that, in a herd handled on range throughout the year, and operated with a short breeding season, Bang's disease should never become a serious problem.

These cattle are normally grazed throughout the year, although they have been fed hay during varying winter periods when the weather was unusually severe, and in seasons of severe drouth. Certain lots of the cattle have been fed cottonseed cake as a supplement to grazing. They are run in large, fenced pastures and do not mix with other cattle. The bulls are turned in with the cows between July 1 and July 5, and removed between August 15 and August 20, with the result that all the calves are born before June 1. A period of one month elapses between the birth of the last calf and the resumption of breeding. The cattle are individually identified by tattoo marks and ear tags, and a complete individual history is available for every cow.

The Bang's disease work with this herd was started in 1931. The plan was to test the entire herd twice a year and to correlate the breeding history of the cows with the serological findings. The test used during the first three years was the standard tube agglutination test, the dilutions starting at 1:25. Since 1935, the whole-blood field agglutination test as developed at this laboratory[1] has been used in conjunction with the tube test, the results of the field test corresponding very closely to the tube test results at the 1:50 dilution.

The management of the herd was continued on the same plan as before. No cattle were eliminated from the herd on the basis of reaction to the agglutination test. The original objective was to determine whether, under the method of management used at the experiment station, the infection could be kept at an insignificant minimum without resorting to the elimination of reactors to the agglutination test. The observations were continued on this basis until 1935, when an outbreak of Bang's disease developed, automatically completing the first phase of this work. The plan was then changed to test the effectiveness of culling reactors to an annual fall test in maintaining a clean herd.

Incidence of Bang's Disease from November 1931 to September 1935

The first herd agglutination test was made in November 1931. For the purposes of this report, the test results recorded refer only to the mature cows of the breeding herd. The yearling and 2-year-old heifers also were tested, but the results are not included in this report. There were 20 reactors at 1:100 in the 287 cows of the breeding herd, a percentage of 7. Twenty-

*Paper No. 132, journal series, Montana State College and agricultural experiment station; a project of the Montana Veterinary Research Laboratory, with the Montana experiment station and Montana Livestock Sanitary Board coöperating.

seven cows reacted at 1:50. All the high-titre reactions occurred in cows at least 4 years old, which indicated that there had been no recent spread of infection, although one positive cow had aborted during the preceding winter.

In June 1932, there were nine (3 per cent) of the breeding herd of 278 cows which reacted at 1:100, and 16 at 1:50, all of which had reacted in 1931. Of 339 cows tested in November, eight (2.4 per cent) reacted at 1:100, and twelve at 1:50, with no new reactions. During the previous winter, there was only one abortion, the aborting cow being negative to the agglutination test. Hay was fed to two thirds of the cows for varying periods, but no spread of infection occurred on the feed grounds.

Again in 1933, there was no evidence of spread of the disease. In June, 325 cows were tested. Eight of the former reactors were still positive at 1:100, and 15 at 1:50. On the October test, three of 386 cows reacted at 1:100 and ten reacted at 1:50. Two abortions had occurred during the winter of 1932-1933, but neither cow reacted. The cattle were grazed all winter.

There was no spread of Bang's disease in the herd between the first test in November 1931 and the test in October 1933. In 1934, however, serological evidence of infection appeared in cows which previously had been negative. In June there were twelve cows out of 324 in the breeding herd which reacted at 1:100. Of these twelve, only two had been reactors on previous tests. The number of reactors at 1:100 disclosed by the October test was the same as in June, although the percentage dropped to 3 because the number of breeding cows increased to 410.

During the preceding winter, 200 of the cows were fed hay. All of the new reactions developed in this hay-fed group, and, although the spread of the infection may be explained on the basis of favorable conditions for transmission in the hay-fed lot, the source of the infection which caused the first new case is not clear. Although six of the old reactor cows remained in the herd, none of them aborted. There were four abortions, two of which occurred in cows which developed positive reactions, but neither of these two had previously been a reactor. Therefore, we can not definitely fix the responsibility for the increase of infection on any one of the known-reactor cows, but the possibility of any outside source of infection is extremely remote.

In 1935, the number of reactors increased sharply. There were 52 (14.6 per cent) complete agglutinations at 1:100 in 355 cows tested in June, while 67 reacted in 1:50. There were eleven abortions and eight stillbirths in that season, all of these 19 cows being reactors except two of those whose calves were born dead. In September, 402 cows were tested, with 45 (11.2 per cent) reacting at 1:100 and 57 (14 per cent) at 1:50. This rapid spread of infection occurred during the winter of 1934-1935, when all of the cattle were fed hay for four months. This change from grazing to hay feeding was made necessary by the shortage of grass caused by the severe drouth of 1934. Although the origin of the outbreak which started during the winter of 1933-1934 is obscure, it seems justifiable to correlate the spread of the disease during the two seasons with the feeding of hay on the ground, as compared with grazing on the range.

As a result of this acute outbreak of Bang's disease, the first phase of the work with this herd was considered completed, with the conclusion that, although the disease might never assume significant proportions under strictly range conditions, the presence of a few reactors in the herd might be responsible for a serious outbreak of the disease when winter hay feeding produced favorable conditions for transmission.

Elimination of Reactors to Test Made in Fall Months

The situation in 1935 presented an opportunity to test the theory that in a range herd of cattle, with a limited breeding season, it should be possible to eliminate Bang's disease by removing all reactor cows found in one test made in the fall. This theory is based on two considerations. One of these is that premise infection is negli-

TABLE I—Results of agglutination tests of breeding cows, correlated with breeding record.

Date of Test	No. Tested	1:100 Reactors		1:50 Reactors		Calf-Crop Percentage	Abortions and Stillbirths		
		No.	%	No.	%		No.	%	No. Reacting
1931 Nov.	286	20	7.0	26	9.0	86	6	3.0	1
1932 June	356	9	2.5	16	4.5	74	4	1.8	1
Nov.	340	8	2.4	12	3.5	..	..	....	..
1933 June	325	9	2.8	15	4.6	86	5	1.7	0
Oct.	386	3	0.8	10	2.6	..	..	....	..
1934 June	324	12	3.7	16	4.9	83	7	2.3	3
Oct.	409	12	3.0	20	4.9	..	..	....	..
1935 June	355	52	15.0	67	19.0	69	19	7.0	17
Sept.	402	45	11.2	57	14.0	..	..	....	..
1936 Sept.	354	10	2.8	10	2.8	79	12	4.5	4
1937 Oct.	307	0	0.0	1	0.3	64	6	3.8	0
1938 Oct.	321	0	0.0	0	0.0	85	6	2.3	0
1939 Oct.	370	0	0.0	0	0.0	92	5	1.7	0

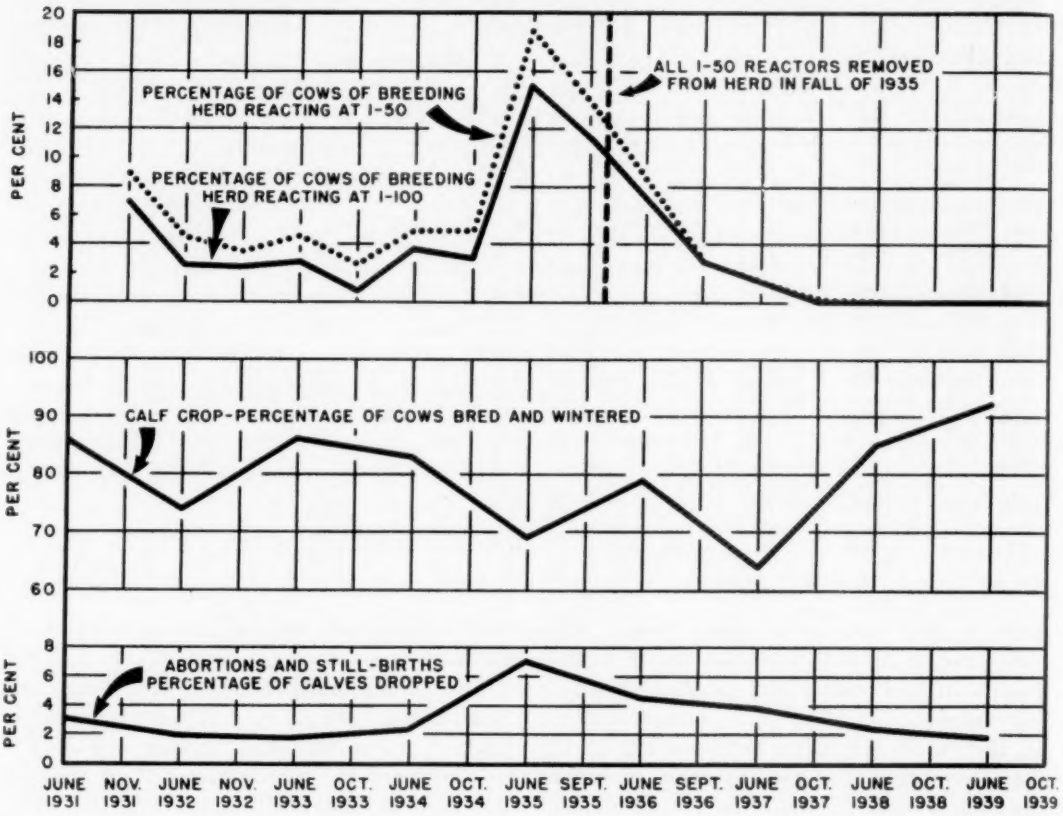
gible in the case of a range herd. The other factor is considered as being fundamental in this connection—a limited breeding season. At the U. S. range station, all the calves are born before June 1. On the assumption that the infection is spread practically only at the time of parturition or abortion, and for a period not to exceed 30 days following parturition, and that nonpregnant cows are only slightly susceptible to infection, no cows should become infected between June 1 and such time as abortions may occur following subsequent breeding. Therefore, if such a herd is tested on October 1, four months have elapsed since there has been opportunity for new infection. During that period any cow which might have acquired infection during the spring should have developed agglutinins. Theoretically, then, it should be possible to eliminate every infected cow as the result of one test made in October.

In the fall of 1935, all reactor cows were shipped. In this case, all animals which showed any agglutination at 1:50 were removed from the herd. This culling process may have been slightly more drastic than we usually recommend for range herds, which is on a basis of complete agglutination at 1:50, this being the titre at which the whole-blood field test selects reactors. Seventy-one reacting cows were shipped from the breeding herd of 402 cows, and with them were culled six cows which had been reactors but had become negative.

Theoretically, this removal of all reacting cows should have eliminated the infection from the herd. In fact, however, the desired result was not realized. During the winter of 1935-1936, five abortions and seven stillbirths occurred, with two of the cows in each of these groups developing a definitely positive agglutination titre. The herd was not tested again until September 1936, when there were ten animals which reacted at 1:100. A possible explanation of the failure to remove all infection from the herd is that one cow, which aborted and reacted in 1935 and was supposed to have been culled from the herd, was recorded as tested again in 1936. However, it is impossible definitely to connect the appearance of reactor cows in 1936 with the reactor cow which was carried over, as there is no record of an abortion charged to this cow during the winter of 1935-1936.

The ten reactors were removed from the herd following the test in September 1936, and the cattle were tested again one year later, in October 1937. On this test there

CHART I



were no reactors at 1:100, but one cow showed complete agglutination at 1:50. This cow and one other which reacted slightly at 1:50 were culled from the herd. Again in October 1938, the breeding herd of 321 cows was tested, with the result that there were no complete reactions at 1:50. In October 1939, 370 cows were tested, with no reactions.

Three successive annual herd tests having produced no positive reactors, it is concluded that the herd is now free from Bang's disease. It is considered that these results constitute strong evidence that the theory upon which this work was based is correct.

Correlation of Serological Findings with Breeding Records

The data discussed above are summarized in table I and chart 1. The three graphs on the chart are plotted on the same time scale. The first graph shows the percentages of reactor cows in the breeding herd for the 13 tests made between November 1931 and October 1939. Several factors are involved in the variations in the curve. The marked drop in reactor percentages between November 1931 and June 1932 was due principally to a decrease in agglutinin titre in a number of the reactors. The slight decreases in reactor percentages in the fall as compared with the previous June tests do not always indicate decreases in actual numbers of reactors, because the number of breeding cows on which the percentage is computed is greater in the fall, as the 2-year-old heifers were added to the breeding herd each year following the June test.

The second graph of the chart shows the calf-crop percentages during the period covered by this study. In general, the calf-crop percentage remained above 80 until

1935, when the lower calf crop coincided with the high point in percentage of reactors, and rose up to 92 per cent in 1939. The low points in 1932 and in 1937 have no relation to Bang's disease, but were due to high percentages of dry cows. This was explained for 1937 by the fact that the breeding season in 1936 was extremely dry, and many cows were never bred. Throughout the entire time, the calf-crop percentage was never seriously affected by abortions, although it is likely that the number of abortions would have been a serious factor in 1936 if the cows which developed reactions in 1935 had remained in the herd.

The third graph shows the percentage of abortions and stillbirths over the eight-year period. This curve closely follows that of the percentages of reacting cows, except that the curve is flatter.

CONCLUSIONS

The data discussed in this report appear to constitute evidence to justify the following conclusions:

1) The presence of a small percentage of reactor cows in a herd which runs on the range throughout the year does not constitute a serious menace.

2) The presence of reactor cows in a range herd may be the source of a serious outbreak of Bang's disease if the cattle are fed hay during the winter.

3) In a range herd where the breeding season is limited so that all the calves are born within a 60-day period, the infection can be eliminated from the herd by shipping all 1:50 reactors to two annual tests made about four months after calving is completed, even in the face of an active outbreak of the disease.

The results with this herd of cattle support the following recommendations for the control of Bang's disease in range cattle, the general principles of which have been recognized for some years in the work of the Montana Livestock Sanitary Board:

1) The breeding season should be limited to not more than 60 days, if possible. This is recognized as good practice from a production standpoint as well as for disease control.

2) All cows should be tested in the fall, three to four months after the last calf was born, and cows reacting with complete agglutination at 1:50 should be culled from the herd. One or more retests may be made at intervals of one year.

3) As an aid in maintaining a clean herd, replacement heifer calves should be vaccinated.

Reference

[1]Welch, H., and Marsh, H.: A whole-blood field agglutination test for Bang's disease in range cattle. Jour. A.V.M.A., lxxxvi (1935), n. s. 39 (4), pp. 493-507.

The Human Teeth

Of the organs of the human body, the teeth are the most vulnerable. They start wearing out as soon as they arrive and cause plenty of trouble throughout all ages: Toothaches, fillings, bridges, plates and whole dentures, often by 40 or before. Few persons have much of a dental apparatus at the Biblical age of 70. No other essential organ gives out as does the teeth.

Animals are more fortunate. Their teeth last through the usual span of life and they cost but little to keep them in working order.

Ariboflavinosis

Keratitis is a specific effect of riboflavin deficiency. Beginning with the conjunctiva the pathological effect reaches the cornea and from there extends to the uvea, whence the visible signs (in man) one sees in equine periodic ophthalmia develop. Other symptoms of ariboflavinosis are cheliosis, glossitis, seborrhea, pruritus and variable degrees of photophobia and impaired vision. As the course of the deficiency proceeds keratitic precipitates occur in the anterior chamber and synechia complicates the picture.

Thiamin chloride, nicotinic acid, cevitamic acid, crystalline vitamin A and cod liver oil make no impression on the disease. On the other hand, cures are quickly obtained by the administration of riboflavin. *(Public Health Reports, January 26, 1940, pp. 157-168.)*

The Effect of Copper Sulfate and Ferric Sulfate on Coccidial Oöcyst Output in Feeder Lambs*

By JOHN F. CHRISTENSEN, Ph.D.
Washington, D. C.

DURING a recent study of feedlot coccidiosis in lambs in central Nebraska, an opportunity was afforded to test the effect of ingestion by the host animals of small amounts of a solution of copper sulfate and ferric sulfate on the level of a natural coccidial infection. This test was suggested by the observation of Spindler[1] that daily administrations of small quantities of a dilute aqueous solution of these chemicals to three spring lambs in Maryland produced a significantly lower average level of oöcyst discharge during the early natural infection than was noted in three control lambs. Each of his test animals was given daily for the first 77 days of the experiment 4 cc. (1 dr.) of a solution made by dissolving 70 Gm. (2.3 oz.) of each chemical in 3,000 cc. (3 qts.) of tap water, and further diluting this basic solution in three parts of tap water before dosage.

Following Spindler's lead, it was desired to determine whether this reduction in oöcyst discharge might be similarly produced in lambs confined to feedlots for fattening, by having available small amounts of these chemicals for daily consumption during the first several weeks of confinement, when clinical coccidiosis is most likely to develop.

MATERIALS AND METHODS

Twenty range lambs from a single shipment from New Mexico were separated into two groups of ten each on the day following their arrival at the Nebraska feedlots. These groups were placed in adjacent pens, each 16 ft. long by 16 ft. wide, made from feedlot panels each composed of three horizontal boards spaced about 6 in. apart. The lambs were able to muzzle each other through these panels, but the feed and water containers were well separated. The pens were located in one corner of an ordinary feedlot used at the time of this experiment as a hospital pen for lambs with severe coccidial scours. The experimental lambs and their controls could thus also contact sick lambs through the panels.

Since the two groups of animals came from the same flock, they were assumed to have equal susceptibility to coccidiosis. In open troughs raised about a foot above the ground, they were fed identical rations consisting of a mixture of high-grade chopped alfalfa, molasses and ground corn. Contamination of feed with fecal material was not difficult and approximated that occurring constantly in normal feedlots. Drinking water was obtained from wells and given fresh daily in open tubs. Conditions of nutrition, water supply, management, susceptibility and opportunities for ingestion of infective oöcysts being the same, it was assumed that the two groups of lambs picked up potentially equal infections and that differences in average oöcyst discharge were produced by the administrations of the chemicals.

The test solution was prepared by dissolving 70 Gm. (2.3 oz.) each of powdered copper sulfate and powdered ferric sulfate in 3 liters (3.15 qts.) of tap water. A cloudy, brownish precipitate formed and settled, and only the clear supernatant fluid was used. From the first day of confinement throughout the duration of the test, 60 cc. (2 oz.) of this solution was added daily to the drinking water of the lambs in one pen, each test lamb thus receiving on the average 6 cc. (1.5 dr.) of the solution daily while the control lambs received none.

Oöcyst counts to measure the natural coccidial infections in the two groups of

*From the zoölogical division of the Bureau of Animal Industry, U. S. Department of Agriculture.

lambs were made about every three days from the beginning of the experiment on November 11, 1939, to its termination on December 17, 1939. Deem and Thorp[2] reported that the usual feedlot infection, as measured by oöcyst discharge, required one or two weeks to rise to a peak and remained high for another three weeks before subsiding. The five-week period covered in the present test, therefore, was sufficient to carry the lambs through most of the interval of maximum oöcyst discharge.

To obtain fecal samples representing an average for the group, two random pellets or their equivalent were obtained from at least seven or eight fecal specimens from each set of ten lambs every time collections were made. Each of these representative samples was placed in a mortar, thoroughly mixed into a paste with tap water, and a small amount of the uniform mixture removed with a pipette to a 15-cc. (0.5 oz.) graduated centrifuge tube. The paste was then washed from the sides of the tube into the bottom by forcibly ejecting 1 cc. (15 minims) of clean water against the inside of the tube near the top, and the resulting suspension allowed to settle. After 30 minutes, the volume of sediment was read to the nearest .05 cc., diluted 14 to 1 with clean water, the suspension thoroughly mixed, and 0.04 cc. of the diluted suspension removed to a slide for counting. Oöcysts were counted and counts calculated and recorded as "oöcysts per cc. of feces." While this method lacked the refinements of usual dilution counting methods in which calculations are determined as "oöcysts per gram of feces," it had advantages in simplicity, convenience and speed which particularly adapted it to the conditions under which this study was conducted.

RESULTS

Figure 1 shows the comparative records of oöcyst discharge for the two groups of lambs. The levels of infection in the two groups were similar from November 11 to November 27, or the first 16 days of confinement inside the enclosures; both groups showed the low-grade oöcyst discharges characteristic of chronic coccidial infections in healthy lambs. Beginning on November 30, however, and throughout the remaining period of the test, the level of oöcyst discharge for the treated lambs was constantly and significantly lower than for the lambs receiving no chemical. The treated lambs remained thrifty throughout the test, showing no digestive disturbances at any time. The untreated lambs, despite a significantly higher average level of oöcyst discharge, remained normal with

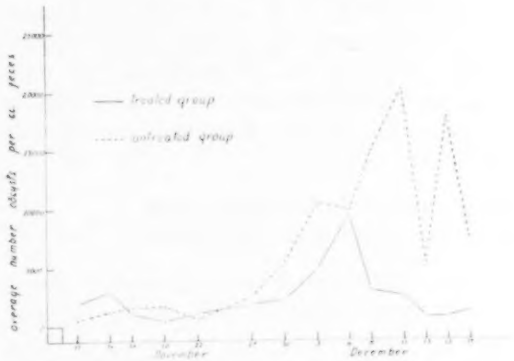


Fig. 1. Comparative records of oöcyst discharge in copper sulfate-ferric sulfate treated lambs and untreated lambs.

one exception. One lamb in this group passed diarrheic feces on December 15, showing a count of 78,000 oöcysts per cc. of feces, although the average count for the entire group including this specimen was 18,000.

CONCLUSIONS

The result of this test is offered only as confirmation of Spindler's[1] observation that the administration of a solution of copper sulfate-ferric sulfate in some manner reduced the average oöcyst output in spring lambs in Maryland, and is not intended to indicate that a similar procedure will effectively control coccidiosis in feeder lambs. It is strongly indicated from these tests, however, that the effect of these chemicals on oöcyst output should be tested with larger numbers of lambs under actual feedlot conditions of sanitation, population and nutrition. Should such experiments demonstrate the same reduction in oöcyst discharge for large lamb populations as was

Studies on the Total Ketone Bodies, Sugar and Calcium of the Blood of Nonpregnant, Nonlactating Ewes*

By JESSE SAMPSON, D.V.M., Ph.D., and L. E. BOLEY,† D.V.M.
Urbana, Ill.

IN THE COURSE of an investigation now in progress at the Ilinois station on certain experimental and clinical aspects of ketosis in cattle and sheep, it semed worth while to make some preliminary observations on the possible effects of such factors as exercise, fasting and diet on the ketone bodies, sugar and calcium of the blood of nonpregnant, nonlactating ewes. The results of these observations form the basis of this report.

PROCEDURE AND METHODS USED FOR THE CHEMICAL ANALYSES

Five nonpregnant ewes of good to choice quality, approximately 1½ years old and weighing an average of 68 kg. (115 lbs.), were used for these studies. Except for the periods of fasting, the ewes were kept in a small dry lot. Feeding was done indoors and the ewes were allowed access to sheltered quarters during bad weather. While on the fasting experiments the animals were confined in quarters with either wood or cement floors. Fresh water was available at all times and loose salt was fed several times each week.

All samples of blood were drawn from the jugular vein. The ewes were subjected to as little excitement as possible before and at the time of bleeding. No attempt was made to withhold the morning feed on those days that samples of blood were collected inasmuch as it was deemed unwise to interfere with the regular schedule of feeding (twice daily except when hay was fed *ad libitum*). If only one sample was collected during the day, the blood was usually drawn between the hours of 8:00 and 9:00 a. m. Potassium oxalate was employed as the anticoagulant for samples analyzed for total ketone bodies and sugar. An effort was made to use uniform amounts of the oxalate. Samples collected for calcium analysis were allowed to clot and the serum removed. The blood filtrates were prepared promptly from the oxalated samples.

The method of Van Slyke and Fitz[1] was used for the quantitative determination of total ketone bodies in whole blood. The suggestion made by Koch[2] that the acetone precipitate be cooled in a desiccator rather than in room air was followed in these analyses. The Van Slyke-Fitz[3] factor of 1.24 was used in calculating the total ketone bodies as acetone per 100 cc. (3.3 oz.) of blood. Blood sugar was determined by the Shaffer-Hartmann-Somogyi method according to the technic described by Koch.[2] In discussing this method, Koch states that "the reducing substances left in the filtrate are said to be true reducing sugars only." Serum calcium was determined by the Clark-Collip[4] modification of the Kramer-Tisdall method.

*From the laboratory of animal pathology and hygiene, University of Illinois; presented before the section on research at the 76th annual meeting of the A.V.M.A., Memphis, Tenn., August 28 to September 1, 1939.

†Assigned by the state department of agriculture to the laboratory of animal pathology and hygiene to assist in diagnosis and research.

(*Continued from preceding page*)

noted here for small numbers, it is possible that the use of a copper sulfate-ferric sulfate mixture, preferably mixed as a powder with feed, would prove to be a valuable supplement to proper nutrition and management in eliminating the ever-present danger of outbreaks of clinical coccidiosis.

References

[1]Spindler, L. A.: Effect of ferric sulphate and copper sulphate on coccidiosis in lambs. No. Amer. Vet., xx (1939), 5, pp. 33-38.

[2]Deem, A. W., and Thorp, F.: Variation in numbers of coccidia in lambs during the feeding season. Vet. Med., xxxiv (1939), 1, pp. 46-47.

TABLE I—Diurnal changes in total ketone bodies, sugar and calcium of blood of nonpregnant ewes.*

TRIAL NO.	HOUR	TOTAL KETONE BODIES†	BLOOD SUGAR MG. PER 100 CC.	SERUM CALCIUM MG. PER 100 CC.	RATION (PER EWE PER DAY)
1	8:30 a.m.		55.14	12.01	Shelled corn, 2 lbs.
	11:30 a.m.		58.31	12.18	Alfalfa hay, 2.5 lbs.
	4:30 p.m.		57.86	12.19	
2	8:30 a.m.		57.18		Shelled corn, 2 lbs.
	10:30 a.m.	0.55	53.79		Alfalfa hay, 2.5 lbs.
	1:30 p.m.		59.21		
	4:30 p.m.	0.94	62.38		
3	8:30 a.m.	0.89	51.07	12.07	Alfalfa hay *ad libitum*
	11:30 a.m.		61.92	12.08	
	4:30 p.m.	1.46	58.99	12.07	
4	10:00 a.m.	3.30	77.71		Shelled corn, 2 lbs.
	5:00 p.m.	2.01	71.64		Alfalfa hay *ad libitum*

*Each value is the average of the determinations for five ewes at the time designated in the table.
†Expressed as acetone, mg. per 100 cc. whole blood.

DIURNAL CHANGES IN TOTAL KETONE BODIES, SUGAR AND CALCIUM

A number of investigators[5, 6, 7] have reported that feeding has no pronounced diurnal effect on the blood sugar of ruminants. Less attention has been given, apparently, to studies of diurnal changes in other constituents of the blood in these species of animals, although Robinson and Huffman[8] and Palmer and Eckles[9] have reported some interesting observations on day to day fluctuations in the level of blood calcium in cattle. These investigators found that significant changes may occur in the calcium content from one day to the next in bovines. Robinson and Huffman[8] state that this fluctuation may be as much as 2 mg. per 100 cc. of blood.

The data collected in the studies reported here failed to reveal any appreciable changes in the total ketone bodies, sugar, and calcium of the blood of nonpregnant, nonlactating ewes for a period of at least eight hours on the days that the trials were conducted. The data for these studies are shown in table I.

EFFECT OF EXERCISE ON BLOOD SUGAR AND SERUM CALCIUM

Allcroft and Strand[7] found that emotional excitement and exercise produced a distinct rise in the blood-sugar level of sheep. The effect on calcium was not studied. We have confirmed their observations. Moderate exercise, in the form of walking for 15 minutes, produced only a slight increase in blood sugar. Vigorous exercise, on the other hand, in the form of forced running for ten minutes, produced a tremendous rise in the blood-sugar content. (See figure 1.) The percentage increase following strenuous exercise was variable, but decidedly significant for all ewes, *viz.*, ewe 1, 148 per cent; ewe 2, 95 per cent; ewe 3, 31 per cent; ewe 4, 73 per cent; and ewe 5, 81 per cent. Ewe 1 was exhausted near the end of the ten-minute period and it seemed doubtful that she would survive. Complete rest resulted in recovery.

These results indicate that the activity of the animals at the time of bleeding may be responsible for a rise in blood sugar. However, if ewes are not chased or otherwise disturbed on the morning the samples are collected, it is not likely that the small amount of activity associated with the act of bleeding is of any consequence. Sheep are tractable animals, and after the routine becomes established, submit to this procedure without undue excitement or struggling.

Neither moderate nor violent exercise resulted in any change of the level of blood calcium (fig. 1).

EFFECT OF FASTING

Two fasting trials of eight- and seven-day periods were conducted, the second four weeks after the first. All of the ewes, from outward appearances, were in a good state of nutrition at the beginning of each fast. The prefasting ration for trial 1 consisted of 2 lbs. of shelled corn and about 2.5 lbs. of alfalfa hay per ewe daily. The prefasting ration for trial 2 was limited to approximately 4.5 lbs. of alfalfa hay per ewe daily.

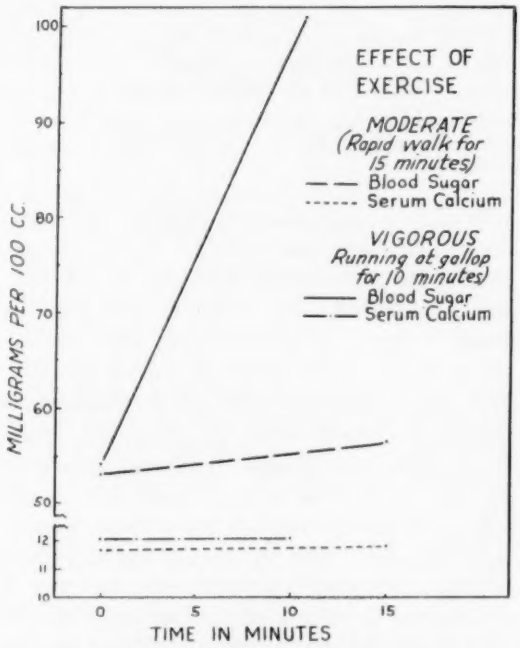


Fig. 1. Effect of moderate and violent exercise on blood sugar and serum calcium. Values represent the averages for the five ewes.

Determinations were made for total ketone bodies, sugar, and calcium of the blood before, during and after each of the fasts.

Clinical Observations.—No definite symptoms of illness were manifested by any of the ewes during either trial, but a characteristic indifference to surroundings and listlessness were observed to develop in all of the animals near the end of each fasting period. The listlessness was especially pronounced in trial 2. Shelled corn was not fed during the first few days following the fast in trial 1, since it was believed that indigestion might develop if the ewes were allowed access to grain before they were consuming the usual amounts of alfalfa hay.

The ewes had excellent appetites when the first trial was completed. Attention is called to this point for the reason that whereas the appetite was apparently unimpaired by trial 1, it was distinctly affected by trial 2; so much so, in fact, that the ewes showed little interest in the alfalfa which was fed at the conclusion of the second fast. Not only did the ewes show little desire for alfalfa but shelled corn failed also to stimulate evidence of hunger. It was clearly evident that the physiological well-being of the animals was lower at the end of the second fast, despite the fact that the ewes had regained most of the weight that had been lost during trial 1 when trial 2 was started. The average loss in weight per ewe in trial 1 was 7.25 kg. (16 lbs.) while the average loss in weight per ewe in trial 2 was 5 kg. (11 lbs.). The obvious difference in the well-being of the ewes at the time each fast was over can probably be attributed to the higher condition of the ewes at the beginning of the fast in trial 1 and to the sustaining effect of a more liberal prefasting ration used in trial 1 as compared with the prefasting ration used in trial 2.[10]

The ewes continued to have a capricious appetite for several days after the conclusion of the fast in trial 2.

Chemical Data.—The chemical data collected for each of the fasting periods will not be discussed separately because the trend of the results was the same for both trials. For the sake of convenience the data for serum calcium, total blood-ketone bodies and blood sugar will be considered in this order.

Serum calcium was not significantly changed by an eight- or seven-day fast. The calcium level appeared to be slightly more constant in trial 2 than in trial 1. The data for calcium are shown in figure 2.

The data collected for total ketone bodies showed that an appreciable though not a great increase occurred in the concentration of these substances. The increase did not develop until the last few days of each fast and was not in any way comparable

with the intense ketonemia that characterizes clinical[11] or experimentally induced[12] ketosis in pregnant ewes. (See figure 2.). Hodgson, Riddell and Hughes[5] reported that a nine-day fast in five dairy heifers produced a rapid decrease of the blood-sugar level. The decrease exceeded 50 per cent of the initial content. Allcroft and Strand[7] studied the effect of fasting on the blood sugar in several sheep and concluded from their results that a seven-day fast in this species was not accompanied by any appreciable change in the level of this constituent. Magee[6] observed a significant decrease in the blood sugar of the fasting goat.

In contrast to the observations of Allcroft and Strand,[7] the results of our studies on the effect of fasting on the blood sugar in nonpregnant, nonlactating ewes were found to be in more general accord with the findings of Hodgson and his associates[5] for cattle and those of Magee[6] for goats, although the decrease was about one half (25 to 30 per cent) as great in ewes as in heifers (fig. 2).

The average value for sugar during the prefasting period in trial 1 was approximately 52 mg. per 100 cc. of blood, while the average value during the prefasting period in trial 2 was about 40 mg. per 100 cc. The level of blood sugar was higher and the fluctuations more marked for the prefasting period in trial 1 than in trial 2, yet the trend was essentially the same for both trials. In these particular experiments there seemed to be a definite relationship betwen the level of the blood sugar during the prefasting periods and the level to which it fell during the intervals of fasting. It is quite possible that the intensity of the hypoglycemia produced during the periods of fasting had a direct bearing on the difference in well-being that was so apparent in all of the ewes at the conclusion of the fast in trial 2 as compared with trial 1. Mention already has been made that the ewes had excellent appetites at the end of trial 1, while partial to complete anorexia was evident at the termination of trial 2.

A gradual rise in the blood-sugar content occurred about the fourth day of each fast. This was probably associated with increased protein catabolism, but no urinary nitrogen determinations were conducted to establish this point.[13]

There was a lag in the rise of the sugar level at the conclusion of the fast in trial 2 and this can be explained by the observation that already has been mentioned, namely, that the ewes ate sparingly when food was made available. An abrupt increase was found on the eleventh and twelfth days of trial 2 four and five days,

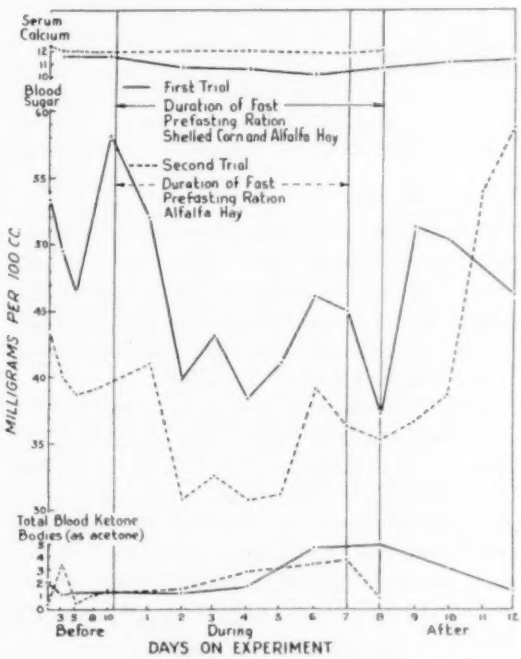


Fig. 2. Effect of fasting on calcium, sugar and ketone bodies of blood. Values represent the averages for the five ewes.

respectively, after the termination of the fast, and can be accounted for by the fact that it was not until the tenth day that the ewes manifested a normal desire for either alfalfa hay or grain. On the twelfth day the average stood at 58 mg. per 100 cc. of blood. The ewes were now cleaning up the feed regularly and, to all outward appearances, had recovered from any adverse effects of the fast. A graphic representation of the data for sugar is shown in figure 2.

TABLE II—Effect of diet on total ketone bodies, sugar and calcium in the blood of nonpregnant ewes.*

RATION (PER EWE PER DAY)	TOTAL KETONE BODIES†	NUMBER OF DETERMINATIONS	BLOOD SUGAR MG. PER 100 CC.	NUMBER OF DETERMINATIONS	SERUM CALCIUM MG. PER 100 CC.	NUMBER OF DETERMINATIONS
I Shelled corn, 2 lbs. Alfalfa hay, 2.5 lbs.	1.44	20	53.30	40	11.63	15
II Alfalfa hay, 3.5 lbs.	1.38	20	40.34	20	12.06	20
III Alfalfa hay *ad libitum*	1.29	10	65.99	20	12.14	10
IV Timothy hay *ad libitum*	0.72	10	55.91	20	12.18	10
V Shelled corn, 2 lbs. Alfalfa hay *ad libitum*	3.24	10	68.98	20	12.29	10

*Each value is the average of the total number of determinations for five ewes.
†Expressed as acetone, mg. per 100 cc. whole blood.

EFFECT OF DIET

The purpose of this phase of the study was to determine the effects of certain feeds and combinations of feeds, fed at different levels for short periods (an average of one week), on the total ketone bodies, sugar and calcium of the blood.

Blood samples were not collected for chemical analysis until the ewes had become accustomed to the ration that was to be fed during the trial. The ewes were group-fed; hence, no attempt was made to determine the amount of feed consumed by each animal. Keen appetites were manifested throughout this series of experiments.

The ration used in trial 1 consisted of 2 lbs. of shelled corn and 2.5 lbs. of alfalfa hay per ewe daily; in trial 2, alfalfa hay alone at the rate of approximately 3.5 lbs. per ewe daily; in trial 3, alfalfa hay *ad libitum;* in trial 4, timothy hay *ad libitum;* and in trial 5, 2 lbs. of shelled corn and alfalfa hay *ad libitum* per ewe daily.

Clinical Data.—None of the rations fed in these trials produced any appreciable change in the level of total ketone bodies. The data for these substances were not studied statistically since the relatively small number of determinations for each experiment did not justify such a procedure. A total of 70 determinations were made for the five trials. The range was from 0.12 mg. to 7.44 mg. per 100 cc., and the average was 1.56 mg. Only two, or less than 3 per cent of the total number of determinations, were values of 5 mg. or more per 100 cc.; three, or approximately 4 per cent, were between 4 to 5 mg.; one, or about 1.4 per cent, between 3 to 4 mg.; eleven, or approximately 16 per cent, between 2 to 3 mg.; 21, an even 30 per cent, were between 1 and 2 mg.; and 32, approximately 46 per cent, less than 1 mg. per 100 cc. of whole blood. A summary of these data is shown in table II.

No significant changes occurred in serum calcium under the conditions that prevailed during these studies. The average for a total of 65 determinations was 12.03 mg. per 100 cc. of serum. (See table II.)

The results of the analyses for sugar clearly demonstrated that the level of this constituent was affected by different methods of feeding. The average value for trial 1 was 53.30 mg. per 100 cc. of blood; for trial 2, 40.34 mg.; for trial 3, 65.99 mg.; for trial 4, 55.91 mg.; and for trial 5, 68.98 mg. per 100 cc. The data for sugar were analyzed statistically. A significant difference was found between the average value for trial 1 and the average for each of the others, except trial 4. The average for trial 2 showed a significant difference

from each of the other four averages. The value for trial 3 showed a significant difference as compared with the averages for trials 1 and 2. There was probably some significance in the difference between the values for trials 3 and 4, but none whatever between the values for 3 and 5. The average value for trial 4 differed significantly from the values for trials 2 and 5. With the exception of the average for trial 3, the value for trial 5 showed a real significant difference from the averages for all other trials. The data for blood sugar are summarized in table II.

SUMMARY

Data have been presented on a study of the influence of a number of factors upon certain constituents of the blood of nonpregnant, nonlactating ewes.

No appreciable differences were found between the respective values for total ketone bodies, sugar and calcium of the blood at 8:30 a. m. and the values for these constituents at 11:30 a. m. and 4:30 p. m.

Moderate exercise produced no change in the calcium level and only a slight increase in the sugar level. (No determinations were made for ketone bodies.) Vigorous exercise did not affect the calcium content but produced a tremendous increase in blood sugar.

Fasts for either eight or seven days produced 1) a definite drop in the sugar content, 2) an appreciable though not a marked increase in the total ketone bodies, and 3) no effect on the calcium level. The increase in ketone bodies was hardly significant when compared with the intense ketonemia found in clinical or experimental (fasting) ketosis in pregnant ewes.

A characteristic listlessness was manifested by all of the ewes during the last few days of each fasting period. Anorexia developed during one of the trials. The possible causes for these manifestations are discussed.

Various rations, fed at different intake levels for short periods (an average of one week), were found to affect very decidedly the blood-sugar content but did not produce any appreciable changes in the concentrations of total ketone bodies or serum calcium.

References

[1]Van Slyke, D. D., and Fitz, R.: Studies of acidosis. VIII. The determination of B-hydroxybutyric acid, acetoacetic acid, and acetone in blood. Jour. Biol. Chem., xxxii (1917), p. 495.

[2]Koch, F. C.: Practical Methods in Biochemistry (Baltimore, 1934).

[3]Peters, J. P., and Van Slyke, D. D.: Quantitative Clinical Chemistry. Vol. 11, Methods (Baltimore, 1932).

[4]Clark, E. P., and Collip, J. B.: A study of the Tisdall method for the determination of blood serum calcium with a suggested modification. Jour. Biol. Chem., lxiii (1925), p. 461.

[5]Hodgson, R. E., Riddell, W. H., and Hughes, J. S.: Factors influencing the blood-sugar level of dairy cattle. Jour. Agr. Res., xliv (1932), p. 357.

[6]Magee, H. E.: Observations on digestion in the ruminant. Jour. Exp. Biol., ix (1932), p. 409.

[7]Allcroft, W. M., and Strand, R.: Studies on the lactic acid, sugar, and inorganic phosphorus of the blood of ruminants. Biochem. Jour., xxvii (1933), p. 512.

[8]Robinson, C. S., and Huffman, C. F.: Studies on the chemical composition of beef blood. Jour. Biol. Chem., lxvii (1926), p. 245.

[9]Palmer, L. S., and Eckles, C. H.: Normal variations in the calcium content of the blood of dairy cattle. Jour. Dairy Sci., xiii (1930), p. 351.

[10]Morgulis S.: Fasting and Undernutrition; a Biological and Sociological Study of Inanition (New York, 1923).

[11]Sampson, J., and Hayden, C. E.: Physiological aspects of ketosis in cows and ewes with special reference to carbohydrate metabolism. Cornell Vet., xxvi (1936), p. 183; also New York State Vet. Coll. Ann. Rpt. (1935-36), pp. 85-99.

[12]Fraser, A. H. H., Godden, W., Snook, L. C., and Thomson, W.: The influence of diet upon ketonemia in pregnant ewes. Jour. Physiol., xciv (1938), p. 346.

[13]Morgulis, S., and Edwards, A. C.: Chemical changes produced in the blood by fasting and subsequent refeeding. Amer. Jour. Physiol., lxviii (1924), p. 477.

Keeping Qualities of Encephalomyelitis Vaccine

It has been shown that encephalomyelitis vaccine (chick embryo) will retain its potency for ten to tweve months. Duplicate samples kept at room temperature for six weeks lost more than one half in antigenicity. Potency was completely lost when the vaccine was stored under the same conditions for more than three months. (*Canadian Journal of Comparative Medicine, iv, February 1940, p. 1.*)

Vitamin E deficiency leads to resorption of the embryo in pregnant females and degenerative changes in the testicles of the male.—*Evans and Burr* (1927).

Ovine Thelaziasis*

By M. A. STEWART, Ph.D.
Davis, Calif.

DURING the past nine years we have become increasingly aware that canine thelaziasis, caused by *Thelazia californiensis* (Price), is not of rare occurrence. According to Douglas,[1] 43 cases have been reported from California. Kofoid and Williams[2] have reported a single case of human thelaziasis caused by the same nematode; this was probably an accidental infestation. The only other host reported as being naturally infested by this parasite is a cat examined by Douglas.[3]

All of the above-mentioned cases have occurred in California, although their distribution clearly indicates the probability that *T. californiensis* occurs in other western states. It has been assumed that this nematode is a natural parasite of the dog and possibly even confined to this host, except for possible accidental infestations, in the adult stage of development.

Up to the present time at least 21 species of parasitic nematodes have been placed in the genus Thelazia. All of these species are viviparous and live in the lacrimal duct, conjunctival sac, eye or nictitating membrane of mammals and birds. Although species of Thelazia are known to infest cattle, horses, camels, swine, dogs and wild mammals and birds in the Old World, no species has been recorded heretofore in sheep. *T. rhodesi* (Desmarest) has been reported from cattle and *T. lacrymalis* (Gurlt) has been reported from horses in North America, but *T. californiensis* is the only known indigenous species attacking domesticated mammals.

On October 21, 1939, the writer found specimens of *T. californiensis* in the eyes of sheep in three distinctly separated flocks in Sonoma county, Calif. On December 28, 1939, specimens of the same species taken from the eyes of a buck deer (*Odocoileus hemionus* subsp.) in Sequoia National Park, Calif., were received from F. R. Oberhansley of the United States National Park Service.

The worms collected from the eyes of sheep were all from flocks suffering from a condition diagnosed as infectious keratitis. During the course of treatment of this disease the owners of the animals accidentally observed the presence of the parasites. From one to twelve worms were collected from the eyes of the sheep examined and about 75 worms were taken from the eyes of the deer. Kofoid and Williams[2] reported that the specimens of *T. californiensis* which they obtained from the human eye were slightly smaller in nearly all measurements than the specimens described by Price.[5] The specimens secured from both sheep and deer, however, did not differ significantly in measurements from those given in the original description. The males of this worm measure 7.7-13 mm. by 0.35-0.5 mm. and the females, 12-18.8 mm. by 0.36-0.53 mm.

The diagnosis of thelaziasis is difficult at times. It depends ultimately, of course, upon finding and identifying the parasites, but these worms only occasionally move across the cornea and pupil. When they do they move across the more readily visible portions of the eyeball, they can be seen easily because of their size and whitish color, but they are difficult to capture and remove, unless immobilized, because they are extremely active and agile in their movements and quickly gain refuge in the more inaccessible regions beneath the membrana nictitans, in the conjunctival sac or lacrimal duct. When these parasites are numerous in the eye they are not difficult to find, but when only a few are present careful and sometimes prolonged search is necessary. The writer has repeatedly examined an eye which he knew to be infested from having caught a glimpse of a worm before seeing the parasite again and obtain-

*From the division of entomology and parasitology, University of California.

ing it. Beneath the membrana nictitans is the most profitable place to look for the organisms.

In view of the difficulties sometimes encountered in finding the parasites, the diagnosis of thelaziasis frequently depends upon recognition of the early symptoms. In the infested sheep examined by the writer excessive lacrimation, engorged blood vessels of the membranes and the eyeball and petechiae of the membranes and beneath the lids were observed. It must be borne in mind, however, that these symptoms may have been due to the infectious keratitis in some of the animals in the bands. One of the owners of the sheep reported having observed a small sinus extending into the conjunctiva. Some of the infested animals manifested no symptoms. In the deer the cornea was slightly opaque, there was some inflammation and the conjunctival tissues were so badly congested that when the eyelids were pulled back this swollen tissue stood out in large bulges.

It has been observed in those mammals in which thelaziasis has been studied that the parasites cause irritation of the conjunctiva which results eventually in a more or less severe conjunctivitis with excessive lacrimation, vascular injection of the conjunctiva, edema of the lids and photophobia. The eyes may remain closed and tenderness may be very acute. If the conjunctivitis is not promptly and adequately treated, complications of keratitis and sometimes purulent ophthalmia may develop. The worms cause distress to their hosts by their presence and movements on the sensitive membranes and the hosts show a general nervous reaction, especially when the parasites are in the vicinity of the inner canthus. Ordinarily, however, the symptoms, particularly the primary ones, are relatively mild and disappear soon after the removal of the parasites.

It has been observed in dogs and cattle infested with *T. californiensis* and *T. rhodesi*, respectively, that the movements of the worms across the cornea result in gradual scarification, with increasing opacity, and may lead to complete blindness if the animals are not properly treated. Vision is apparently impaired in direct proportion to the length of time the parasites are present in the eye or the frequency of reinfestation. Probably, however, no serious impairment of vision is likely to occur unless the infestation remains untreated for several months, or the magnitude of infestation is great or reinfestation occurs frequently and repeatedly. Douglas[1] reported that one dog became permanently blind from thelaziasis, and cattle infested with *T. rhodesi* in Africa have become blind and also have shown other severe pathological changes, including the secondary invasion by suppurative organisms, which usually results in complete destruction of the eyeball. Udrinski and Cuirea (reported in Price[5]) recorded a bovine infestation with *T. rhodesi* in which, in addition to photophobia and epiphora, there was a chronic keratitis accompanied by atrophy of the eyeball.

In a camel infested with *T. leesei* (Railliet and Henry) a cyst the size of a pea was observed on the inner surface of the membrana nictitans. Eight adult worms were discovered in the liquid contents of this cyst. The host appeared to suffer no ill effects.

Howard,[6] in recording an infestation of humans with *T. callipaeda* (Railliet and Henry), reported that the presence of these parasites in the conjunctival sac is accompanied by intense pain and gives rise to extreme nervous symptoms. In one case he found larvae in an advanced stage of development attached to the epithelial layer of a wart-like papilloma of the lower eyelid. Trimble[7] believes that the presence of adult *T. callipaeda* in humans may possibly be responsible for paralysis of the muscles of the lower lid and cause ectropion.

Inasmuch as these various species of Thelazia all behave in much the same manner and since all become localized in the region of the eye, it may be safely assumed that the pathogenesis in sheep and deer infested with *T. californiensis* is about the same as that in the various mammalian hosts infested with the several Thelazia species mentioned above.

The treatment of thelaziasis has received relatively little attention and, obviously, such attention has been directed toward the

treatment of dogs, cattle, horses and camels. The most common therapeutic practice is to desensitize the eye by the instillation of a few drops of cocaine, or some other suitable local anesthetic, thereby permitting adequate exploration, and then to remove the worms with forceps or a small cotton swab. Not only does cocainization allow thorough examination but it will also cause at least many of the worms to crawl out of the inner canthus.

Although cocaine is usually the preferred local anesthetic for use in the eye, certain facts must be borne in mind, particularly when its repeated employment may be indicated, as in the treatment of thelaziasis. The prolonged or repeated use of this anesthetic in the eye will produce by virtue of its continued action a haziness in the superficial cells of the cornea, especially when combined with the employment of precipitating antiseptics, such as mercuric chloride. Rather than the 2 per cent solution of cocaine that is usually recommended, it is better to make several applications, if necessary, of a 1 per cent solution. Novocaine is slower in action and, therefore, not as popular as cocaine, but it does not produce the drying and injury to the superficial corneal epithelium that frequently follows the use of cocaine. A 10 per cent solution of novocaine does not produce mydriasis or disturbance of accommodations and it does not increase intraocular pressure.

When there is reason to believe that all of the parasites have not been removed from the folds of the conjunctiva, an antiseptic collyrium, such as 1 per cent cresol, a 1:2,000 mercuric chloride solution or a solution of boric acid, may be applied daily for several days, following which the worms may be removed by means of forceps or irrigation. The mercuric chloride solution is contraindicated, as described above, when cocaine is being employed.

After treatment consists of washing the eye frequently with boric acid solution, a 1:4,000 solution of potassium permanganate, or a 1:4,000 solution of mercuric chloride, as in simple conjunctivitis, until the inflammation has subsided.

In those cases where the parasites may become lodged in the anterior chamber of the eye, as has been reported in horses infested with *T. lacrymalis*, paracentesis of the cornea should be performed. The consequent escape of the aqueous humor usually brings with it the parasites. Sometimes, however, it is difficult to determine whether or not the worms have been expelled with the fluid. Usually the parasites are found on the sleeve of the operator and, consequently, it has been suggested that a black coat should be worn in such cases. If the worms have not escaped, they must be removed with fine forceps introduced through the corneal wound.

Recovery is almost always rapid and uneventful, but several examinations should be made after the initial removal of the parasites in order to determine definitely whether or not all of them have been recovered.

It is perfectly obvious that effective prophylaxis is dependent upon a complete knowledge of the life history of the parasite. Unfortunately, practically nothing is known of this life history. Thus far only adult worms have been found in the eye. The eggs close to the genital pore in gravid females have thin, membranous shells and contain fully developed coiled larvae. These eggs are forced out in large numbers when the parasites are placed in physiological saline.

Direct transplantations made by Kofoid, Williams and Veale[4] of adult *T. californiensis* were only partially successful; the worms survived for but a few days. Faust[8] transplanted adult *T. callipaeda* into the conjunctival sac of a dog and rabbits and they were still alive at the end of a year. He also had some success with transplantations into the eyes of cats and a monkey, but he was unable to make successful transplantations into the eyes of a goat and a sheep. However, in each case there was a decrease in the number of worms instilled into the conjunctival sacs, which indicated that they died off gradually. Direct ocular instillation of larval *T. callipaeda* into dogs failed. The possibility of natural direct transmission of thelaziasis seems un-

likely also because of the relatively large numbers of worms present in many cases.

Since an eyeworm of chickens, *Oxyspirura mansoni* (Cobbold), which is closely related to the genus Thelazia, is known to require a cockroach, *Pycnoscelus surinamensis* (Linn.), as an intermediate host, it has been commonly supposed that the Thelazia species probably have arthropod intermediate hosts. Kofoid, Williams and Veale[4] failed to infect cockroaches with larvae of *T. californiensis* and Faust[8] was unable to infect various species of "filth flies," dog flies (*Hippobosca francilloni* [Leach]) and cockroaches (*Blatella germanica* [Linn.]) with larvae of *T. callipaeda*. None of these experiments, however, eliminates the possibility that thelaziasis is transmitted to mammals by an arthropod intermediate host.

It seems likely to the writer that the gravid females or their eggs may escape directly from the eye or, more probably, perhaps *via* the nasolacrimal duct to the ground or vegetation and, there, be subsequently ingested by an appropriate intermediate arthropodan host, which is, in turn, ingested by a susceptible definitive mammalian host. An abundance of such possible intermediate hosts exists and they should be thoroughly investigated. It is, of course, possible that the eggs hatch in the region of the eye and that the larvae make their way into the peripheral circulation where they are picked up by an intermediate blood-sucking arthropodan host, but, in the opinion of the author, non-blood-sucking arthropods are to be suspected first.

Kofoid, Williams and Veale[4] believe that canine infestation with *T. californiensis* occurs in the late summer and fall, and Faust[8] has noted a similar seasonal distribution of *T. callipaeda* in dogs in China. Douglas,[1] however, reported that the majority of cases of canine thelaziasis caused by *T. californiensis* come to the attention of practicing veterinarians in the winter and spring. The cases in sheep and deer reported in this paper were observed in the autumn. One of the infested bands of sheep was introduced into Sonoma county three weeks before the parasites were noted and at that time had no apparent eye trouble, which may mean that the infestation was picked up within three weeks before adult worms appeared in the eyes.

Douglas[1] states that in every case of canine infestation with *T. californiensis* where a history was available, the dog had either lived in a wooded rural region or had been known to visit such areas frequently. In several cases the dogs had been employed in hunting or working domestic animals. The sheep observed to be infested with this parasite were all in a hilly region well wooded and with a lot of brush and, obviously, the infested deer in Sequoia park also picked up the parasites in a wild region.

It is interesting to note that none of the dogs on any of the ranches where infested sheep were found showed any evidence of thelaziasis.

Conclusions

The discovery of *Thelazia californiensis* in abundant numbers in the eyes of sheep and deer in widely separated areas, and particularly the absence of infestation in dogs closely associated with infested sheep, strongly suggests that this worm is normally a parasite of herbivorous mammals and only accidentally a parasite of dogs, as it is of man. It is quite likely that it will be picked up in cattle and possibly also in horses.

The association of thelaziasis and infectious keratitis was probably merely a coincidence, but it is possible that the worms may act as a predisposing factor in the latter infection.

References

[1]Douglas, J. R.: A survey of canine thelaziasis in California. Jour. A.V.M.A., xciii (1938), n.s. 46 (6), pp. 382-384.

[2]Kofoid, C. A., and Williams, O. L.: The nematode *Thelazia californiensis* as a parasite of the eye of man in California. Arch. Ophthal., xiii (1935), pp. 176-180.

[3]Douglas, J. R.: The domestic cat, a new host for *Thelazia californiensis* Price, 1930 (Nematoda: Thelaziidae). Proc. Helminth. Soc. Washington, vi (1939), p. 104.

[4]Kofoid, C. A., Williams, O. L., and Veale, N. C.: *Thelazia californiensis*, a nematode eye worm of dog and man, with a review of the thelazias of domestic animals. Univ. California Publ. Zoöl., xli (1937), pp. 76-86.

[5]Price, E. W.: A new nematode parasitic in the

The Susceptibility of Guinea Pigs to Equine Encephalomyelitis Virus Inoculated Through Various Routes*

By CARL F. SCHLOTTHAUER, D.V.M.
Rochester, Minn.

VARIOUS sporadic enzoötic and epizoötic nervous diseases are known to occur among horses. The symptoms and pathological characteristics of some of these diseases are similar. Since the widespread occurrence of the disease designated by Haring, Howarth and Meyer[1] as encephalomyelitis, there is a tendency to classify all nervous diseases of horses that are manifested by general symptoms and occur during the spring, summer or fall as equine encephalomyelitis. This practice, if continued, will lead to error in evaluating our present methods of vaccination and treatment to prevent or cure this disease.

In 1931, Meyer, Haring and Howitt[2] demonstrated that equine encephalomyelitis is a specific disease of horses and mules and that it is caused by a specific virus. At present two distinct types of viruses producing a similar disease in horses and mules are known to occur in North America. These are designated eastern and western type. The latter is the more widespread, but the former is more virulent.

Meyer, Haring and Howitt[2] were the first to demonstrate that equine encephalomyelitis could be transmitted experimentally to various laboratory animals, as well as horses, by intracerebral inoculation with brain tissue from animals spontaneously infected with this disease. Similar results have been obtained by other workers. It is generally recognized that intracerebral inoculation of an adequate dose of equine encephalomyelitis virus will produce encephalitis in approximately 100 per cent of susceptible animals so inoculated. When other sites are used for inoculation, a varying incidence of infection occurs. Shahan and Giltner[3] experimentally transmitted equine encephalomyelitis to horses and guinea pigs by intracutaneous, intramuscular and subcutaneous inoculation of virus and by rubbing it into abrasions in the mucosa of the tongue. These methods produced varying results. Vawter and Records[4] transmitted equine encephalomyelitis to a horse by instilling the virus intranasally. Intraperitoneal inoculation likewise gives varying results.

In my experiments I sought to learn the reliability of the various methods of inoculation into susceptible test animals of infective material from cases of equine encephalomyelitis. This was done, first, to provide a standard to evaluate results of vaccination or other prophylactic treatment and, second, to establish a standard whereby one can more accurately evaluate negative results following the inoculation of suspected material into experimental animals through various routes for diagnostic purposes.

EXPERIMENTAL PROCEDURE

Guinea pigs were selected as the experimental test animals. Inoculations of 20 per cent suspensions of fresh infective brain tissue in physiological saline solution were made intracerebrally, intralingually, subcutaneously, intraperitoneally and intracutaneously into the foot pad of the left hind foot and the hairless tip of the nose. The guinea pigs were inoculated in groups of three or more. This was repeated until

*From the division of experimental medicine, The Mayo Foundation; presented at the 76th annual meeting of the A.V.M.A., Memphis, Tenn., August 28 to September 1, 1939.

(*Continued from preceding page*)

eyes of dogs in the United States. No. Amer. Vet., xii (1931), pp. 49-58.

[6]Howard, H. J.: Thelaziasis of the eye and its adnexa in man. Amer. Jour. Ophthal., x (1927), pp. 807-809.

[7]Trimble, C. G.: Circumocular filariasis. China Med. Jour., xxxi (1917), p. 266.

[8]Faust, E. C.: Studies on *Thelazia callipaeda* Railliet and Henry 1910. Jour. Parasitol., xv (1928), pp. 76-86.

TABLE I—Results of inoculation with 0.2 cc. of 20 per cent brain suspension in physiological saline solution.

Site of Inoculation	Number of Guinea Pigs	Died			Incubation Periods (Days)	Survived	
		Encephalitis		Other Causes		Immune	Susceptible
		Number	Per Cent				
Intracerebral	18	18	100.0		4–5		
Foot pad (L. hind)	18	13	72.2	1	5–7	4	
Intracutaneous (nose)	18	8	44.4		5–8*	10	
Subcutaneous	18	8	44.4	1	5–10	8	1
Intraperitoneal	18	8	44.4	1	5–9	8	1
Intralingual	18	8	44.4	2	5–9	7	1
Contact controls	9	1 (18 days)	1.1	1	18		7

*One guinea pig succumbed on the 14th day.

18 guinea pigs had been used to test the efficacy of each site of inoculation.

Federal bureau of animal industry Iowa (western) equine encephalomyelitis virus was used in all of the experiments. The minimal lethal dose of the brain suspension was not determined. But before these experiments were begun, it was learned that 0.1 cc. (1.5 minims) of a similar brain suspension injected intracerebrally into a susceptible guinea pig always produced fatal encephalitis. To insure that an adequate quantity of virus was always present, not less than 0.2 cc. (3 minims) of brain suspension was used to inoculate each guinea pig in these experiments. Fresh suspensions were prepared from fresh guinea pig brains each time immediately before the animals were inoculated. The virulence of each brain suspension was determined by intracerebral inoculation into three or more guinea pigs. All of the guinea pigs were anesthetized with ether to facilitate inoculation. Those that survived the initial inoculation received 0.2 cc. of brain suspension intracerebrally after two weeks, to test them for immunity. The brains of all of the guinea pigs that succumbed were studied microscopically to determine the presence of encephalitis.

Results

Table I reveals that intracutaneous inoculation into the pad of the left hind foot produced encephalitis in 72 per cent of the guinea pigs so inoculated, whereas only 44 per cent of those receiving a similar inoculation into the skin of the nose succumbed. It will be noted that the efficacy of inoculation into the skin of the nose and by the subcutaneous, intraperitoneal and intralingual routes was identical in so far as infecting the animals was concerned. However, one of those surviving in each of the groups inoculated subcutaneously, intraperitoneally and intralingually failed to acquire sufficient immunity to withstand an intracerebral inoculation of 0.2 cc. of infective brain suspension two weeks following the primary inoculation.

All of the guinea pigs receiving intracerebral inoculations of 0.1 cc. or more of 20 per cent brain suspension succumbed from encephalitis on the fourth or fifth day, but a marked variation in the virulence of different lots of brain suspensions was noted when they were inoculated intracutaneously, subcutaneously, intraperitoneally and intralingually. Great care was exercised in making the inoculations to insure uniform technic. It is my opinion that the variation in results can not be attributed to differences in technic.

Kelser[5] mentioned that transmission of the virus of equine encephalomyelitis by mosquitoes occurs after multiplication, maturation or, less probably, certain cyclic changes of the virus within the mosquito. It is possible that the virus does undergo a process of maturation or some cyclic change within the donor or recipient which might account for the variation in results when brain suspensions are inoculated in sites other than the brain. Similar variations in the virulence of different lots of brain suspension were noted when 1 cc. (15 min-

ims) was inoculated subcutaneously and intraperitoneally.

The virus of equine encephalomyelitis differs markedly from that of rabies as regards its incubation periods when inoculations are made in various sites. As will be noted in table I, guinea pigs inoculated intracutaneously in the pad of the left hind foot succumbed to encephalitis as rapidly as those inoculated intralingually. King[6] observed that the virus of eastern equine encephalomyelitis when injected peripherally invades the blood stream and passes directly from the blood into the brain.

All of the susceptible guinea pigs receiving intracerebral inoculations succumbed to encephalitis. Microscopic study of the brains from these animals revealed diffuse encephalitis identical with that observed in the brains of those which succumbed to encephalitis following inoculation in other sites. The lesion in the cerebrum caused by the needle tends to produce only a localized reaction and is rapidly repaired. A series of guinea pigs were inoculated intracerebrally with 0.2 cc. of a 20 per cent suspension of brain containing virus. Some were killed and their brains were removed for microscopic study after 24, 48, 72, 96 and 120 hours. In all cases it was observed that the encephalitis remained localized until after 72 hours, or until symptoms of encephalitis were manifested.

CONCLUSION

These experiments indicate that intracerebral inoculation of material suspected of containing equine encephalomyelitis virus is more reliable than inoculations made in other sites when guinea pigs are used as the test animals.

References

[1]Haring, C. M., Howarth, J. A., and Meyer, K. F.: An infectious brain disease of horses and mules (encephalomyelitis). No. Amer. Vet., xii (Oct. 1931), pp. 29-36.

[2]Meyer, K. F., Haring, C. M., and Howitt, B.: Newer knowledge of the neurotropic virus infections of the horse. Jour. A.V.M.A., lxxix (1931), n. s. 32 (3), pp. 376-389.

[3]Shahan, M. S., and Giltner, L. T.: Some aspects of infection and immunity in equine encephalomyelitis. Jour. A.V.M.A., lxxxiv (1934), n. s. 37 (6), pp. 928-934.

[4]Vawter, L. R., and Records, E.: Respiratory infection in equine encephalomyelitis. Sci., lxxviii (1933), pp. 41-42.

[5]Kelser, R. A.: Transmission of the virus of equine encephalomyelitis by *Aedes taeniorhynchus*. Jour. A.V.M.A., xcii (1938), n. s. 45 (2), pp. 195-203.

[6]King, L. S.: Studies on eastern equine encephalomyelitis. II. Pathogenesis of the disease in the guinea pig. Jour. Exp. Med., lxix (May 1, 1939), pp. 675-689.

Public Health Personnel

Of the 211 cities in the United States with a population over 50,000, about one fourth have trained men directing their sanitary programs. The majority of the inspectors in municipal health departments have had no educational training on public health. *(American Journal of Public Health, xxx, January 1940, p. 85.)*

Susceptibility of Guinea Pigs to Trichinosis

The guinea pig appears to be a much more reliable experimental host in trichinosis research than do mice, rats, swine or rabbits.

The minimum infection dose to cause the death of a guinea pig was 800 larvae. A dose of 2,000 larvae killed 32 out of 34 guinea pigs during the fourth to eighth week after infection, *i.e.*, the acute muscular stage. Massive doses of 12,500 and 18,750 larvae were fatal even in the first week after infection during the acute intestinal stage. *(H. Roth. Experimental Studies on the Course of Trichina Infection in Guinea Pigs. II. Natural Susceptibility of the Guinea Pig to Experimental Trichina Infection. American Journal of Hygiene, section D, xxix, 1939, pp. 89-104.)*

Schumacker and Heuser of the department of poultry husbandry, Cornell University, have demonstrated that riboflavin, the G component of vitamin B, improves the hatchability of eggs.

Nonius Horses and Puli Dogs

By JOHN R. MOHLER, V.M.D., D.Sc.
Washington, D. C.

TWO STALLIONS and four mares of the Nonius breed were imported in November 1935 from Budapest, Hungary, by the federal bureau of animal industry at the request of Secretary Wallace, who has personally done much work in the field of genetics. These horses are now being used in certain breeding investigations conducted at the U. S. Range Livestock Experiment Station at Miles City, Mont. They were selected by Adolph Eichhorn, director of the federal animal disease station at Beltsville, Md., during one of his European trips. As a number of members of the American Veterinary Medical Association have written inquiring about this little-known breed of horses, as well as the slightly better-known Puli dog, the following information may be of interest.

The Nonius is a horse found principally in the Danubian countries, especially Hungary, Jugoslavia, and Rumania. It is essentially a half-bred horse, having developed from matings of the foundation sire "Nonius Senior," a French stallion born in 1810 whose sire was an English Thoroughbred and the dam a Norman mare. "Nonius Senior" was in the cavalry of Normandy in 1814, when he was captured by the army of Austria-Hungary. The following year he was sent to the Hungarian government stud at Mezöhogyes. He and his male get were bred to mares of many breeds (Polish, Russian, Turkish, Arabian, Hungarian, English, Spanish, Mecklenburg, Holstein, and others).

There are two types of Nonius, one large and the other small. The original separation into large and small types was made in 1870 by Count von Horvath, who divided the animals on the basis of actual measurements. Today the distinction is more theoretical than real, many of the small Nonius animals in Hungary having large progeny, and many of the large horses small progeny. Moreover, the distinction is founded partly on the great variability of the original material and partly on the desire to maintain two types. It is said that the large Nonius is especially adapted to large, extensively cultivated farms in the lowlands of Hungary, Jugoslavia and Rumania, and is an excellent artillery horse. The small Nonius, on the other hand, is said to be ideal for the small farmer in the Danubian lands and can be used as a riding or wagon horse. (See figure 1.)

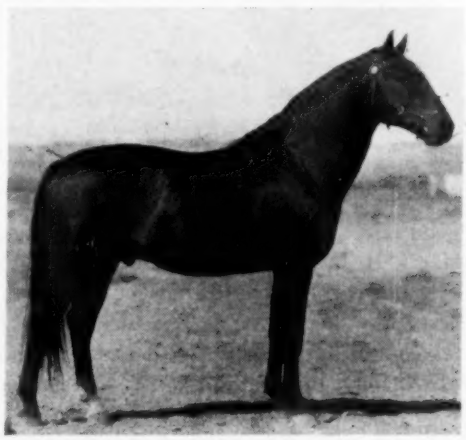


Fig. 1. Nonius stallion "Shorty," foaled in 1932 and imported from Hungary in November 1935.

In general, it may be said that the Nonius is a strongboned, medium heavy, working horse, whose color varies from chestnut to dark brown or black. Sorrels occur as exceptions. Exteriorly it resembles the Norman plow horse, known in France as the "Bourdon."

The variability of Nonius horses in conformation, together with their good disposition, makes them suitable for many uses. The gait is elastic and high stepping. Another characteristic of this breed is the blunt nose. As a rule, they measure about 15.2 to 16 hands.

*From the Bureau of Animal Industry, U. S. Department of Agriculture.

Puli Dogs

On the trip above mentioned, Dr. Eichhorn also brought with him two male and two female Puli Shepherd dogs of Hungary for the bureau's investigations of the intelligence of certain breeds of dogs, particularly with reference to their ability and suitability for farm and ranch purposes in this country.

Those who have studied the characteristics of this breed agree that the Puli is of Nordic origin and has been part of the life

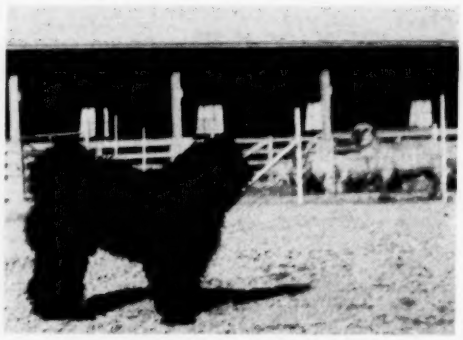


Fig. 2. "Tarcsai Bifkic" (kennel name "Pal"), male, whelped in 1935, imported from Hungary during same year.

of the Hungarian sheep grower for centuries. It is versatile, easily trained and quick on its feet, and has been called a retrieving sheep dog. The body is relatively small and stocky, with the legs moderately long. (See figure 2.) The ears are broad at the base, pointed toward the end and, unlike a number of the other herding breeds, are of the pendant type. The characteristic tail is relatively long, well covered with hair, and is usually carried partially curled over the back. The Puli's coat is rather long and of fine texture. In young dogs the hair appears quite woolly and is very dense. In both mature and immature dogs the coat is wavy and the color is usually black, although some are gray or creamy white. Most of the dogs have a covering of hair over the face, the amount, however, not being as extensive as in breeds such as the Old English Sheepdog.

In temperament they are quite lively and their dispositions are usually very good. From our experience, it is characteristic for animals of the breed to be friendly and affectionate, and they are very intelligent. The height of the animals varies between 15 and 20 inches at the shoulder, and the weight approximates 30 pounds in the male and 23 pounds in the female. Among the outstanding characteristics of the breed are the dense, wavy coat, the active, alert temperament and the well-developed feet and legs.

Who Wrote Shakespeare's Plays?

Although the electromagnetic wave we call x-ray is used for many purposes in the arts and sciences, no one could have predicted that it would be used some day to settle the everlasting argument over the authorship of the Shakespearean classics.

By means of x-ray studies of three famous paintings supposedly of the Bard of Avon, an expert photographer and inquisitive historian* discovered that the paintings are pictures of the Earl of Oxford, who is pointed out by certain groups as the actual author of Shakespeare's plays.

Our "Anti's" Seem to Kill Folks

Among the principal nations of the world, the United States is tops in the incidence of smallpox. Our anti-vaccinationists break all records for their influence, if not also for their numbers.

During the same year that Germany had no cases of smallpox among its 67,000,000 people, and but twelve cases occurred in England and Wales with their 42,000,000 folks, the United States came along with 11,693 reported cases. France with its 40,000,000 population had 273 cases. In 1938, our population of 120,000,000 suffered from 15,000, or nearly 4,000 cases more than the year before (1937).

A medical journal from which we take these figures says the situation is "humiliating," which we think is quite a condescending participle for a row of corpses that long.

---

*Charles W. Barrell, American secretary of the Shakespeare Fellowship.

Factors Limiting Swine Production*

By B. M. LYON,† V.M.D.
Pearl River, N. Y.

THE IMPORTANCE of the swine industry can best be emphasized by a statement in relation to the position the industry holds in the number of animals produced annually in the United States solely for food consumption and their market value. According to figures compiled by the U. S. Department of Agriculture, since 1900 there have been approximately 2,300,000,000 swine produced, with a total value of $24,750,000,000. This represents an income to the swine producers of approximately $650,000,000 annually for the past 38 years.

Considerable fluctuation occurs from year to year, both as to number and unit value. For example, during 1923 there were 69,300,000 produced with a value of $851,745,000 and in 1935 there were 37 million with a value of $236,000,000. During this period, the lowest price was $4.10 per head in 1934 and the highest price per head, $23.25 in 1919. As with all basic commodities, the industry is governed by supply and demand, notwithstanding man-made attempts to standardize or regulate prices by organized slaughter.

The hog-raising industry has become so highly specialized in many sections of the country that it may be divided into breeders and feeders. True enough, a high percentage of our small producers and many of our larger producers are both breeders as well as feeders. However, many of our largest producers breed no pigs whatever, but depend entirely upon shipping feeder pigs from breeding areas to their feedlots, not so much in the grain belt, but more especially in the garbage-feeding areas. While such a specialization, which requires the transportation of large numbers of feeder pigs, has many advantages, it is not without hazards, some of which are of grave concern today and will be spoken of later under disease control.

The success of the individual swine producer is dependent primarily upon his knowledge of the fundamentals of breeding, feeding, and disease control.

ERRORS IN BREEDING

Breeding errors are numerous and the first factor limiting swine production, in the order of occurrence, is a lack of the basic knowledge of the fundamentals of breeding. Selective breeding as to type should be made to meet the requirements of the butcher, feeder and breeder. Breeders may have a preference fancy, but due consideration must be given not only to the most profitable type as to breed, but to type within the breed which will meet the greatest market demand. The wide, short-bodied, short-legged hog, showing extreme refinement of bone, head and hair, so popular 25 years ago, no longer meets the market demand because of high percentage of fat. The meaty, not the fat type of hog, is the one meeting with high favor today.

The mistake of inbreeding is a common one. In many sections the breeding stock has been diminished and any gilt capable of breeding is selected, regardless of its relation to the herd boar. Outcross pigs are much to be preferred to those inbred.

In many localities pasture breeding and group breeding are common practices. Under such circumstances herd boars have been noted to make ten to twelve services a day. Such services result in the production of reduced and weak litters. Observation has shown that litters from sows so bred average two to three pigs, whereas litters produced by proper servicing average eight to ten pigs. Breeding diseased sows is another potential source of weak litters as well as the continual use of herd boars which have proved themselves consistently productive of weak offspring.

*Presented at the annual meeting of the Southern Veterinary Medical Association, Columbia, S. Car., November 9-11, 1939.

†Lederle Laboratories, Inc.

All such breeding fallacies are major factors in increasing the mortality of baby pigs. When it is considered that the loss of baby pigs in the country is shown to be 40 per cent before they reach the age of 30 days, it behooves us to give serious consideration toward reducing these losses.

FEEDING

Another major contributing factor toward baby-pig losses is improper feeding as well as housing. It is a generally accepted procedure, but too frequently disregarded, that a sow's ration should be reduced 50 per cent two to three days prior to farrowing, and a limited ration continued for a week or ten days following. Such a practice limits milk production and aids in giving the pig a proper start. Full feeding just prior to and immediately following farrowing is largely responsible for pig scours, which exacts its toll of baby pigs annually and is chiefly a matter of diet.

Proper housing is also essential to reduce baby-pig losses. Pigs should not be confined to concrete pens but must have access to Mother Earth to avoid anemia and other deficiency diseases. Institution herds are among the common sufferers from this condition. The writer, while posting pigs from an institution herd recently, observed that anemia was so pronounced that the blood resembled a serous fluid or light wine rather than blood, and more than 50 per cent of the pigs died annually as a direct result of a diet lacking in copper and iron.

Food rations, both of the sow as well as the pigs, must contain an adequate supply of calcium and phosphorus to avoid rickets and build the skeleton structure. These essentials may be secured from natural food sources, when given access to Mother Earth, and not necessarily from expensive mineral concentrates so frequently recommended.

Pastures are the foundation of profitable pork production—so important that they will frequently determine the difference between profit and loss. Good pastures, such as alfalfa, clover, and rape, not only provide an excellent and economical food source, but likewise provide sufficient area for proper exercise so essential to good breeding records. Closely confined sows tend to become too fat and will not give as good account of themselves at farrowing time as those forced to take plenty of exercise.

Under proper conditions of feeding, breeding, and care, a fair percentage of sows should produce two litters annually. It is not to be expected that all sows will produce two litters, but from ten sows it is reasonable to expect 15 litters annually. There is no good reason why sows should work only one half of the year.

DISEASE FACTORS

Infectious disease reaps the greatest toll among swine, of any one factor. It is not within the scope of this paper to do more than to treat briefly some of the more important diseases which annually exact a huge toll.

Until recent years, cholera was the only disease which was seriously considered as affecting the swine industry. While cholera still remains paramount of all swine diseases, new infections have made their appearances. Other diseases which it is believed have existed for a generation have more recently become recognized. It seems one of Mother Nature's own provisions that the more intensified or highly specialized the industry becomes, either in the animal or plant field, the more diseases of a destructive nature come into existence. Thus, we have had appear swine influenza, swine dysentery, brucellosis of swine, erysipelas, hemorrhagic septicemia, infectious enteritis, and many important parasitic diseases.

CHOLERA AND ITS CONTROL

Cholera, as previously stated, still remains the most important disease of swine and no large-scale producer would hope to escape the cholera scourge without systematic vaccination. Cholera control, with the use of serum and virus, needs no introduction. There is no longer any question as to the value of the simultaneous method of vaccination. Pigs in good physical condition, properly treated with an active

virus and a potent serum, should be and are rendered immune to cholera and remain so for life. Immediately you take issue and ask what about the reported failures, the so-termed serum and virus breaks, etc.

First of all, permit me to state that serum and virus as produced by the leading manufacturers today are comparatively uniform products of a high standard of efficiency. True, some variation exists and some serums are superior to others, but, on the whole, as produced today, under quite rigid inspection, it is relatively a uniform product. Far greater variation and more irregularities occur in the condition of pigs and the circumstances under which pigs are vaccinated than that which exists in the serum and virus.

Successful immunization is primarily dependent upon two factors: First, a good, active virus balanced with a serum of high potency, properly administered in adequate dosage; secondly, the health and condition of the pig at the time of vaccination and its subsequent care and handling for a week to ten days following vaccination.

In considering vaccination, the first thought in the mind of the average individual is to secure a good, potent serum. To be sure, a good serum is of the utmost importance but, most certainly, it is secondary to the necessity for using a good, active virus. A virus properly selected for its power to immunize, as indicated by its virulency and freedom from contamination, is of paramount importance. In the use of the simultaneous treatment, it must be borne in mind that in the first and last analysis it is the virus and not the serum which is responsible for the immunity production. The serum functions in the capacity of producing a temporary protection, thereby permitting the virus to activate or induce a permanent or lifetime immunity. The immunity or degree of protection thus induced is largely in direct ratio to the virulence or activity of the virus used.

To obtain such a virus, the first responsibility rests with the manufacturer to use the utmost care in its selection and subsequent care and handling while in his possession, as well as to emphasize sufficiently the necessity for proper care after it has left the jurisdiction of the producer. The virus should at all times be kept at refrigerator temperature, approximately 40° to 50° F., and at no time exposed to sunlight or high temperatures. It must be borne in mind that the virus is a living disease agent removed from its host, and to remain active it must be properly handled and stored. Serum should likewise be stored at low temperature, but failure to do so is much less disastrous than failure to store the virus properly. Successful vaccination is likewise dependent upon the serum and virus being administered by or under the jurisdiction of a competent veterinarian familiar with swine and swine diseases. Many difficulties have arisen where virus, as well as other such products, has fallen into the hands of those not qualified to administer or handle it.

The second factor upon which so much is dependent for successful vaccination is the condition of the animals. The responsibility here lies with the herd owner and the veterinarian—the herd owner to the extent of supplying an honest and complete history, and the veterinarian to the extent of his ability to recognize those conditions which are potential sources of post-vaccination hazards.

It is too frequently observed that the herd owner takes great delight in withholding valuable herd history at the time of vaccination, which, if furnished the veterinarian, would have been valuable in preventing otherwise unfavorable results. Careful observation and examination of the herd for the presence of other infectious or debilitating diseases will preclude many complications which arise following vaccination. All too frequently, vaccination receives credit for conditions which develop coincident with, but not as a direct result of, treatment. The existence of anemia, parasitism, infectious enteritis, paratyphoid and pulmonary infection is a potential source of post-vaccinal complications. Where such conditions exist, not only are they exaggerated by vaccination but cholera itself may develop in individ-

uals most severely affected due to their inability to immunize under such circumstances. It is, therefore, important that competent veterinary supervision be given.

Proper care of the pigs following vaccination, both as to handling and feeding, is extremely important. It is not surprising that many feeder pigs shipped under crowded conditions and subjected to severe exposure within 24 hours following vaccination with serum and virus, are lost soon after arrival. Neither is it surprising that heavy losses frequently occur where shipper pigs are vaccinated two to three days following arrival. It is extremely unfortunate that it is not a shipping requirement that all such feeder pigs be vaccinated two or three weeks prior to shipment. Such a procedure would materially reduce or eliminate losses from cholera in shipping feeder pigs. Pigs should likewise be fed very lightly for a few days following vaccination, and under no circumstances gorged with corn or any other feed for at least a week following. One of the surest means of creating post-vaccinal troubles is to overfeed immediately after vaccination.

Briefly, in considering cholera control with serum and virus, let us be fair in our judgment when trouble arises. First, consider that hogs may be and are successfully immunized against cholera. Secondly, other diseases exist and are increasingly prevalent among both vaccinated and unvaccinated hogs under modern conditions of intensive production. Third, differential diagnosis is frequently quite difficult when based on clinical observation only. Fourth, three parties, not one alone, are concerned when trouble occurs, one or all of which may be responsible; namely, the producer of the serum and virus, the veterinarian, and the herd owner.

If the veterinarian exercises due precaution, making a careful observation prior to vaccination, and finds the hogs healthy, he is not at fault when losses occur. On the other hand, if he fails to observe or recognize the conditions prior to vaccination, he may be responsible for losses.

Should the herd owner fail to give a full herd history and fail to carry out the veterinarian's instructions following vaccination, he may be responsible. On the other hand, should the producer fail to deliver to the practitioner a good, active virus, together with a potent serum, he may be at fault. Too frequently, however, the sole defendant is the serum producer, and a good product, specific in its action against cholera only, becomes discredited because it is not a cure-all for complicating diseases or is used under circumstances recognized as potential sources of disaster.

Under disease-limiting factors, I would like to discuss briefly a few of the more prevalent diseases, aside from cholera, which are of ever-increasing importance to the swine industry.

Before doing so, I wish to state that I fully appreciate the position of the practitioner. It is comparatively easy for us to describe or diagnose the typical case of most any disease, such as our text books describe and which we seldom see, but as the busy practitioner daily encounters the atypical and complicated disease symptomatology, it requires all the skill and intuition of the best diagnostician. This is especially true in veterinary medicine, where we receive little aid from our patients.

It is for this very reason that it becomes doubly imperative that we, as veterinarians, avail ourselves of all our diagnostic aids. I refer particularly to diagnostic laboratory service, since in many instances a positive diagnosis can be determined only by skillfully combining the history, symptoms and laboratory findings.

SWINE ERYSIPELAS

More recently, swine erysipelas has been gaining prominence as a disease factor in many hog-raising districts of the United States. In some European countries, the disease is of greater concern than hog cholera.

History and Distribution.—In 1886, Theobald Smith first isolated the causative organism in the United States and, for many years thereafter, it was referred to as diamond skin disease, a mild form of the disease. The acute septicemic form was

not recognized until about 1930, and by 1933 a survey indicated its presence in 17 of the 48 states, chiefly those states having a dense swine population. Since that time, many additional states have identified the disease in both the acute as well as the chronic form.

The disease agent *Erysipelothrix rhusiopathiae* is infectious for other species of animals and fowls, as well as man, but never appears in an enzoötic or epizoötic nature. A number of veterinarians and laboratory workers have become infected.

The infection is most prevalent in the late summer and fall and is most frequently observed in pigs from 50 to 150 lbs., although swine of all ages may become affected.

The mortality has been reported by observers from various sections as averaging from 10 to 25 per cent, and it is said that an additional 10 to 25 per cent of infected herds become chronically affected and of little value.

Course and Symptoms.—The course of the disease varies in different herds and within the same herd from an acute septicemia to a chronic form. Individual hogs may experience either the acute or chronic form, or both.

The symptomatology to an experienced observer is contrasting to cholera and other acute swine infections, but may readily be confused. It is not within the scope of this paper to give a complete clinical and a differential picture.

In distinguishing swine erysipelas from cholera symptoms and lesions, of some help when present are: Stiff gait; arched back; evidence of pain when handled; high average temperature; less mental depression or surprising amount of animation when disturbed; partial retention of appetite; early red discoloration of skin, disappearing upon pressure; infiltration or gangrenous area of skin; arthritis; gross involvement of the spleen; engorged liver; and paint-brush hemorrhage of gastrointestinal serosa.

Serological and blood diagnostic tests have been used to some extent but have limited usefulness. Positive blood reactions are usually absent in the acute stage and, when present, may denote a previous infection.

The administration of specific anti-swine-erysipelas serum, within the first two to three days of illness, brings such marked improvement within 24 to 48 hours in the uncomplicated disease that it is frequently used as a diagnostic aid.

A clinical differentiation of acute swine erysipelas from the acute septicemia type of suipestifer or paratyphoid infection, is frequently impossible.

INFECTIOUS ENTERITIS

Another of our most serious disease factors today, particularly in our most congested hog-raising districts, is infectious enteritis, referred to under varied nomenclature, such as necro, necrotic enteritis, paratyphoid, swine typhus, and salmonellosis.

The *Salmonella suipestifer* is quite generally accepted as the causative organism. Other members of the Salmonella group, such as the paratyphoid, are sometimes involved, but the suipestifer is mainly incriminated, and prior to the discovery that cholera is a filtrable virus, this organism was termed *B. cholera suis* and believed to be the true cause of cholera.

The acute form of enteritis may be produced by feeding the culture but not by injection into the tissue. The infection is commonly associated with cholera and other swine diseases, but alone is capable of producing a primary severe acute infection with a high mortality.

The clinical picture is variegated, dependent upon the form, whether acute or chronic, and the very nature of the infection implies its symptomatology and pathology.

When infectious enteritis appears, it usually affects a fair percentage of the herd. The onset is less noticeable to the herd owner than cholera. Affected animals continue to feed some and show less depression. Constipation is noted in the

early stages, usually followed by a pronounced dysentery. There may be a marked discoloration of the skin of the abdomen which persists under pressure. Mortality is greatest within the first few days of infection, and then a more chronic form is assumed.

Proper feeding, care and sanitation are the best methods of combating this disease. As Dr. Van Es has stated, removal of the cause is the most essential procedure.

A common practice of many is to treat with from one to three doses of bacterin before placing new pigs in the feedlot. Various methods of treatment are used. Withholding grains and placing on a buttermilk diet, or alkalized oat and barley mixtures, is practiced by many. One ounce of powdered copper sulfate in 25 gallons of clear water is suggested by some. Regardless of the method of treatment employed, sanitation is of primary importance, and no treatment can succeed without it.

HEMORRHAGIC SEPTICEMIA

Investigators and authorities differ in their views as to the significance of *Pasteurella suisepticus* infection, some considering it as a primary disease entity, others as a secondary or complicating disease agent only.

To support the primary disease theory, I should like to relate an experience of comparatively recent date. In a herd of approximately 1,000 hogs, fed garbage on an open field, some 200 animals were lost within a few days. I was asked to investigate. The herd was within a few miles of our laboratories and every laboratory facility for aid in diagnosis was available. The animals affected were cholera-immune hogs and upon autopsy showed no indication of cholera. As far as could be determined, some form of septicemia was present. Blood examination showed pure cultures of Pasteurella, and inoculation into susceptible rabbits produced death overnight, from which again pure cultures of the organism were recovered.

The neighboring farmer was a poultry raiser and his chickens grazed over the same garbage feedlot. On visiting the farm the following day, the poultryman advised that he was finding eight to ten dead chickens each morning. Upon laboratory examination of the chickens, the same Pasteurella species was isolated. In this instance, not only was the hemorrhagic septicemia shown to be the primary disease entity responsible for the losses, but the same organism also was responsible for the losses in the poultry flock and the source of the infection was a common one.

There are many lesser disease-contributing factors limiting swine production. However, within the scope of this paper we can but name a few of the more common, such as swine influenza, brucellosis, pig scours, deficiency diseases, parasitism, etc., which annually take their toll of our swine population.

SANITATION

Lastly, but by no means of least importance, is the lack or total disregard of sanitation which exists among the majority engaged in the swine industry. There seems to exist the serious misconception that swine and sanitation are two widely separate entities, and that sanitation may be totally disregarded in so far as swine raising is concerned. There is probably no greater contributing factor to the promotion and dissemination of swine diseases than this prevailing failure to exercise even the first principles of sanitation among swine breeders as well as feeders.

In the final analysis, the factors resulting in limitation of swine production, as well as their control, rest with the breeders and feeders, the practicing veterinarian, and the laboratory. Only by the combined efforts of all responsible parties can we hope to arrive at a reduction of the factors resulting in losses of constantly increasing proportion. Each and all parties concerned must be ready to assume their honest share of responsibility and refrain from "passing the buck," as it were, and in so doing we may arrive at or rapidly further the solution of many of our present problems yet unsolved.

An Attempt to Demonstrate a Filtrable Form of Brucella Abortus*

By C. P. FITCH, D.V.M., M.S., D.Sc., and A. G. KARLSON, B.S., D.V.M.
St. Paul, Minn.

SARNOWIEC[1] recently reported that a filtrable form of *Brucella abortus* may be obtained by growing the microörganism in the presence of tuberculin. Week-old broth cultures containing 10 to 20 per cent of tuberculin were passed through Chamberland L_2, L_3, and L_5 filters. Guinea pigs were injected subcutaneously and intraperitoneally with 1 to 2 cc. of filtrate from each filter. Specific agglutinins and lesions could be demonstrated in the animals inoculated with filtrates from the L_2 and L_3 filters 12 to 15 days following the inoculation. Filtrates from the L_5 filters produced no change in the guinea pigs. A microörganism whose colony characters were those of *Br. abortus* could be isolated from the filtrates (not specified from which filters) as well as from the lesions in the guinea pigs. It failed to grow on subcultures, but when reinoculated into guinea pigs, specific agglutinins were produced.

We have attempted to repeat these results, but we have been unable to demonstrate any filter-passing form of *Br. abortus*.

METHODS

A culture of *Br. abortus* isolated from a guinea pig injected with milk was inoculated into tubes of beef-infusion broth to which had been added phenol-free tuberculin* in amounts of 10, 15 and 20 per cent. Beef-infusion-broth cultures without tuberculin also were made. These were incubated at 37° C. in an atmosphere of 10 per cent CO_2 for one week.

Transfers to serum agar were then made from each medium. Two guinea pigs were inoculated with 0.5 cc. of each of the three cultures containing the various amounts of tuberculin and the culture without tuberculin.

Ten cc. of each culture was then passed through Berkefeld N, Chamberland L_2, L_3, L_5, and L_7 filters, respectively. Two guinea pigs were inoculated with 0.5 cc. each of filtrate from each filter, as shown in table I.

TABLE I—Results of inoculating guinea pigs with filtrates of cultures of Br. abortus in broth containing tuberculin.

Filtrate	Culture			
	Plain Broth	Broth Plus 10% Tuberculin	Broth Plus 15% Tuberculin	Broth Plus 20% Tuberculin
(Unfiltered)	+	+	+	+
Berkefeld N	—	—	—	—
Chamberland L_2	—	—	—	—
Chamberland L_3	—	—	—	—
Chamberland L_5	—	—	—	—
Chamberland L_7	—	—	—	—

+ = *Br. abortus* and specific agglutinin demonstrated in guinea pigs.
— = *Br. abortus* or specific agglutinin not demonstrated in guinea pigs.

Transfers from each of the broth cultures showed growth of *Br. abortus* in two days. Cultures made of the filtrates from each of the five filters were negative after 14 days of incubation under 10 per cent CO_2. All of the guinea pigs were bled by heart puncture two weeks following inoculation. Those injected with unfiltered cultures showed agglutinins specific for *Br. abortus*, with titres ranging from 1:25 to 1:50. The serums of guinea pigs inoculated with filtrates were negative. Three weeks following inoculation all of the guinea pigs were

*From the division of veterinary medicine, Minnesota Agricultural Experiment Station; paper No. 1774, scientific journal series.

*Kindly furnished by R. M. Chapin, chief of the biochemic division, Bureau of Animal Industry, U. S. Department of Agriculture.

destroyed and their spleens were cultured. *Br. abortus* was isolated from all of those receiving unfiltered material, but it was not isolated from any of the animals which had received filtrates. The results are given in table I.

CONCLUSION

We have been unable to demonstrate *Brucella abortus* in filtrates of cultures of the microörganism in broth containing tuberculin.

Reference

[1]Sarnowiec, W.: Sur l'apparition des formes filtrables de *Brucella abortus* (*Brucella bovis*) dans des milieux additionnes de tuberculin. Compt. Rend. Soc. Biol., cxxix (1938), 25, pp. 129-132.

Disease in Wartime

While politicians (diplomats over there) are debating about finance, munitions, and axes, men of medicine are wondering if they know just what they are doing, for every war of history has touched off the torch of pestilence and disease. Though the old scourges, like smallpox, plague, cholera, typhoid, typhus, and syphilis, yield to available preventives, influenza is yet to be conquered. In 1918, it swept the world of over 10 million souls, and is no doubt waiting right now to repeat the performance.

"Five Days of the Best Entertainment Ever Offered Veterinarians' Wives"

When the subcommittee on ladies' entertainment, headed by Mrs. Clifton D. Lowe, met in the national capital recently, plans were made that all point to "five days of the best entertainment ever offered veterinarians' wives." Throughout the session, which will be held at the Mayflower Hotel in Washington, D. C., August 26-30, this important group will be in action to provide a constant good time for every lady in attendance. One of the many features will be a boat trip to Mount Vernon.

SUBCOMMITTEE ON LADIES' ENTERTAINMENT, 77TH ANNUAL MEETING

Seated (left to right): Mrs. Adolph Eichhorn, Mrs. Irving M. Cashell, Mrs. C. D. Lowe (chairman), Mrs. W. Taylor Miller, and Mrs. H. W. Schoening. Standing (left to right): Mrs. Willard H. Wright, Mrs. H. M. O'Rear, Mrs. Walter J. Hall, Mrs. Mark Welsh, Mrs. R. A. Kelser, and Mrs. D. I. Skidmore. (Absentees on account of "flu" are Mrs. George W. Gillie, Mrs. Wm. A. Hooker, Mrs. John R. Mohler and Mrs. Mason Weadon.)

The Occurrence of a Gram-Positive Coccus in Birds Positive to the Serum Plate Test for Pullorum Disease*

By ROSS H. HURT, B.S., D.V.M., and ERNEST C. McCULLOCH, D.V.M., M.A., Ph.D.
Pullman, Wash.

THE POULTRY FLOCK at the agricultural experiment station of The State College of Washington has been blood tested for pullorum disease and the reactors removed for several years. The incidence of reactors has been low. In February 1938, the flock was tested by the whole-blood rapid plate method and between 1 and 2 per cent of the birds reacted. These were removed. A single addition of chicks from a U. S. pullorum-clean flock was made following the test.

In January 1939, pullorum tests were made on 289 hens and pullets in order that pullorum-positive birds could be removed before the beginning of the hatching season. The test was conducted by two technicians who used the serum plate technic and two federal-approved antigens. The samples were drawn from the wing vein and tested in the veterinary laboratory at the poultry plant. Approximately 59 per cent of the group of 121 New Hampshire Reds gave reactions in dilutions of 1:100 and 1:200. Of a group of 27 Rhode Island Reds 26 per cent gave reactions in the same dilutions, and 17 per cent of a group of 141 White Leghorns gave similar reactions. Of the reactions approximately 75 per cent were complete and the other 25 per cent were incomplete or suspicious. The samples were then checked by a third technician using still another federal-approved antigen, his results showing close correlation. The group of reactors were removed and isolated from the rest of the flock.

In checking the results of the test in February 1939, the group was re-bled and parallel tests made by the rapid whole-blood plate technic and the laboratory serum plate method. Interesting variations were noted in these two series of tests. In the first place, the whole-blood rapid plate test did not bear out the serum plate test in all cases. The technic used in testing these samples was that recommended by the companies producing the antigens, *i.e.*, a loopful of blood and a drop of antigen as delivered by the dropper supplied with the antigen. It was found that many more of the birds would give reactions and bear out the findings obtained with the serum plate test when a drop of whole blood as delivered by a dropper pipette was used instead of the loopful with a drop of stained antigen. This would indicate that the titres of the serums were low.

In March and April 1939, small groups of the reactor birds were retested and at each retest the findings by the serum plate method substantiated the previous serum plate tests while the whole-blood tests gave the same results as before. In the March test a group of 37 reactor New Hampshire Red hens gave 13 complete and two incomplete reactions to the whole-blood plate test and 30 complete, three partial, and four nearly complete reactions to the serum plate test. In April another group of reactors were retested and 26 birds gave three complete and four incomplete reactions to the whole-blood plate test but, when tested with the serum plate technic, gave 23 complete and three incomplete reactions in dilutions of approximately 1:500 and 1:100, and 19 complete, four incomplete and three negative reactions in a dilution of approximately 1:200.

In the course of the study, birds were autopsied and cultures taken in an effort to substantiate the findings from the test (tables I and II). The ages of these reactors varied from a year to 2½ years. In one hen a few ovules appeared slightly misshapen though not discolored and the rest appeared normal. In the other birds no sign of lesions commonly attributed to

*Published as scientific paper No. 448, College of Agriculture and agricultural experiment station, The State College of Washington.

Salmonella pullorum was found, the organs and tissues appearing quite normal. Cultures were made from the ovary, heart, liver, spleen and bone marrow. From none of these birds, which gave positive reactions when tested by the serum plate technic, was *S. pullorum* or any other related Gram-negative organism isolated, while a Gram-positive coccus was consistently isolated.

TABLE I—Cultures of Gram-positive coccus from reactors.

BREED	ORGANS CULTURED					NO. BIRDS CULTURED
	H.	L.	O.	S.	B. M.	
W.Leg.	1	1	5	0	3	8
N.H.R.	2	3	15	2	6	18
R.I.R.	1	1	3	0	2	6

W. Leg. = White Leghorn; N. H. R. = New Hampshire Red; R. I. R. = Rhode Island Red.
H = Heart; L = Liver; O = Ovary; S = Spleen; B.M. = Bone Marrow.

REVIEW OF LITERATURE

Hinshaw and Dunlap,[1] in 1927, isolated a Gram-positive coccus from chickens which gave reactions on carbohydrates similar to the reactions exhibited by the coccus isolated at this time. The coccus which they reported appeared as a contaminant in phenolized serums which had been shipped to the laboratory and grew in the samples as well as in the serological tubes in the laboratory. These samples were being taken by the "nicking" method commonly used in taking blood samples in the field. The authors isolated the organism from the skin of the chickens but, failing to isolate it from the heart blood, concluded that the skin was the natural habitat of the organism. They advised the use of an improved technic in taking samples to remove the possibility of contamination of samples, and also suggested the use of the rapid plate method of testing, which would not allow time for the growth of the organism in the samples or of the colonies in the serological tubes in the laboratory.

Johnson and Anderson,[2] in 1936, in a discussion of pullorum disease in turkeys, noted a marked cross agglutination in low dilutions between a Gram-positive coccus (designated in the paper as P_1) isolated from a group of poults, and *S. pullorum*. The coccus was isolated in pure culture from the heart, liver and yolk sacs. They found it to be nonpathogenic for chicks and poults. They reported:

> In running agglutination tests with this organism (P_1) as antigen, we found that the sera of the birds agglutinated P_1 in higher dilutions than they did *S. pullorum*. These facts indicate that there is a marked cross-agglutination with this non-pathogenic organism which might, on the basis of the test, lead to the removal of outstanding breeders that harbor non-pathogenic organisms.

FINDINGS IN CHICKS

When the hatching season got well under way, many chicks which died shortly after hatching were supplied for autopsy from reacting as well as from nonreacting groups of birds. The eggs from reacting birds were hatched in incubators separate from the eggs from nonreacting groups. Cultures from the chicks of the reacting group yielded, in 90 per cent of the cases, pure cultures of the Gram-positive coccus shown by laboratory test to be identical with the organism isolated from the breeding birds, while cultures from chicks of the nonreacting group yielded the coccus in approximately 10 per cent of the cases. Cultures from the chicks included heart, liver and yolk sacs. The same organism has been isolated from chicks from Oregon, Washington and Idaho and from chicks hatched from eggs imported from Canada.

All of the routine sanitary precautions were taken in hatching the chicks. The organism has been isolated from chicks of age groups up to 4 weeks. It is interesting to note that the majority of birds so affected showed a definite omphalitis. Although the incidence of omphalitis was greater in chicks of reacting groups, complete records of the true occurrence of omphalitis are not available; thus, further work is being undertaken to ascertain the true relation, if any, of the organism to the condition. Mortality rates in the birds of reacting groups were higher than the

rates in the chicks from nonreacting groups.

The organism also was isolated from the chicks of a reactor hen which, when brought into the laboratory and autopsied, yielded pure cultures of the organism from the ovary as well as from a fully developed egg, ready for laying, obtained from the oviduct.

THE ORGANISM

The organism was isolated from the birds, their eggs, their chicks and from other chicks on Difco nutrient agar (incubated at 37° C.).

Surface Growth.—Scanty, lenticulate, pin point, glistening, discreet colonies, growth slow, colonies distinct in 48 hours.

Stab.—Surface more abundant, growth, papillate.

Nutrient Broth.—Abundant, flocculent, compact sediment.

Morphology and Staining.—Spheres, average diameter 1.5 μ, occurring singly, diploid, or as tetrads. Stains readily with anilin dyes and is positive to the Gram's stain.

Physiology.—Aerobe and facultative anaerobe. Acid and no gas in nutrient broth (Difco) enriched with 0.5 per cent dextrose, maltose, lactose, saccharose, mannitol, levulose, galactose or dextrin. No acid or gas in nutrient broth (Difco) enriched with 0.5 per cent xylose or dulcitol.

VIRULENCE AND ANTIGENIC STUDIES

In an attempt to check the virulence and antigenic properties of the coccus, a few simple tests were made. In view of the inadequacy and inconclusiveness of the initial work, more intensive tests are now under way.

On March 1, 1939, eight pullets were injected with four cultures of the coccus. The birds were shown to be negative for pullorum disease by the rapid whole-blood test checked by the tube test. The injections were made from saline suspensions of 48-hour cultures shown by examination after Gram's stain to be uncontaminated. The birds were injected intramuscularly and intradermally. Three other birds were kept as controls in the same pen with the injected birds. All birds gave negative results to the rapid whole-blood test and tube test for pullorum disease when retested on the 20th and 27th of March, and showed no sign of disease.

On the 25th of March, 60 one-week-old chicks were divided into four groups and injected with saline suspensions of nine cultures of the coccus. Injections were made intramuscularly in one group, intradermally in the second, perorally in the third, and the fourth constituted the control group. The chicks were brooded under standard-type commercial brooders. The different groups showed no ill effects from the injections or from the organisms even when held to 3 months of age and were shown to be negative to the pullorum test at a later period.

TABLE II—Cultures of Gram-positive coccus from non-reactors.

BREED	ORGANS CULTURED					NO. BIRDS CULTURED
	H.	L.	O.	S.	B. M.	
W. Leg.	0	0	2	0	0	6
N.H.R.	0	1	2	0	1	10
R.I.R.	0	0	1	0	0	5

In the laboratory two young rabbits (does) were injected with formalinized cultures of Gram-positive coccus intravenously. Injections were made on alternate days until five injections had been made. Twelve days later blood was withdrawn from the heart for testing purposes. Blood from other rabbits previously injected with the formalinized cultures of *S. pullorum* and subsequently shown to have high titres when tested with pullorum antigen was used in an effort to demonstrate cross agglutination, but all attempts have failed thus far.

Later, on July 10, 1939, a group of seven birds which had reacted to the previous tests were re-bled and the serum sent to another experiment station for testing with pullorum antigen. At that time the serum of these birds, although giving slight reactions, failed to give reactions typical of those commonly attributed to pullorum infection.

SUMMARY

A Gram-positive coccus was isolated from birds whose serums gave reactions to the serum plate test for pullorum disease. In

the test approximately 59 per cent of a group of 121 New Hampshire Reds gave reactions in dilutions of 1:100 and 1:200. Of a group of 27 Rhode Island Reds 26 per cent gave reactions in the same dilutions, and 17 per cent of a group of 141 White Leghorns gave similar reactions. Of these reactions approximately 75 per cent were complete and the other 25 per cent were incomplete or suspicious. *Salmonella pullorum* or any related Gram-negative organism could not be isolated from these birds. It was found that in this case the rapid whole-blood test did not substantiate the rapid serum plate findings in all cases. The isolation of the coccus from the various organs, as shown in tables I and II, and from the eggs and chicks of reactor hens suggests the possibility of transmission through the egg. The majority of the chicks showed a definite omphalitis.

Further work is being carried on to ascertain the true virulence of the organism for chicks and poults, the mode of transmission and the degree to which experimentally infected birds will react to the various tests for pullorum infection.

References

[1]Hinshaw, W. R., and Dunlap, G. L.: Atypical *Salmonella pullorum* agglutinations caused by bacterial contamination. Jour. A.V.M.A., lxxii (1927), n. s. 25 (5), pp. 594-598.

[2]Johnson, E. P., and Anderson, G. W.: Pullorum disease in turkeys with special reference to fowl typhoid in turkeys. Jour. A.V.M.A., lxxxviii (1936), n. s. 41 (3), pp. 375-384.

Select Needs from Menu

The World's Poultry Congress audience was told by F. N. Marcellus of the Ontario Agricultural College that chickens possess the canny instinct of consuming more cod liver oil from the fountain when the weather is dull and cloudy than during periods of sunshine. Quoting: "When cod liver oil was given in fountains to young chicks and adult birds, they consumed a quantity in proportion to the amount of sunlight available, and at any time when there occurred a period of several days of dull, cloudy weather, the intake of the oil was increased until the weather cleared. The intake would drop to a level below that preceding the cloudy period."

The author pointed out that while free-choice feeding should not be adopted as a routine practice, there are numerous illustrations of this category showing where research should be directed in solving some of the poultryman's vital problems.—*From the Poultry Tribune, February 1940.*

Sulfur in Poultry Practice

Though sulfur given internally was once frowned upon as an insectifuge, recent observations made at the Florida experiment station indicate that feeding 5 per cent of sulfur in mash for a period of three weeks aids in the control of lice, mites and ticks. Moreover, treating the soil of the poultry yard, the dropping boards, litter and nest material with sprinklings of sulfur likewise aids in the control of these parasites.

For sulfurizing the yards about 800 pounds of commercial flowers of sulfur per acre is required. The sulfur is raked into the top soil but it requires about two months to obtain the maximum effect on parasite life. As the effect lasts for a year or more, only an annual sulfurization is necessary. The acidity of the soil thus produced can be neutralized with lime in event the land is to be used for cropping. The measure is recommended particularly where chickens are confined in small yards.

Potassium Chloride in Hay Fever

Observations made at Emory University (Georgia) appear to prove that hay fever and asthma are due to potassium deficiency. Since the distribution of potassium is a function of the adrenal glands, it is to these glands one must look for the cause of these allergic diseases. The curing of 35 cases of asthma and hay fever by means of continuous daily dosing with potassium chloride is the basis of the conclusion.

The body temperature of the heron is 105.8°; of the duck, 109.1°; and of the swift, 111.2°.—*Science and Discovery.*

The Inheritance of Predisposition to Renal Calculi in the Dalmatian*

By CLYDE E. KEELER, Sc.D.
Philadelphia, Pa.

THE RESEARCHES to be described constitute a joint project of Harry C. Trimble of the department of biochemistry, Harvard medical school, and the writer. Most of the work was done at the Harvard medical school, with Dr. Trimble handling the chemical analyses and the writer directing the genetic experiments.

In the metabolic degradation of the urines most mammals change the greater part of them through a uric acid stage ($C_5H_4N_4O_3$) into allantoin ($C_4H_6N_4O_3$). This rule holds in general for all species as we ascend the evolutionary scale until we pass the monkeys. When we reach the apes we find that the last step is omitted almost completely and that most waste products of urine metabolism are excreted in the form of uric acid. Man takes his cue from the apes and does likewise, stopping his urine metabolism at the uric acid stage instead of carrying it on over into allantoin.

But there is one exception to this rule—the purebred Dalmatian Coach Hound. In this respect the Dalmatian breed leaps completely out of the taxonomic order of the carnivores over the ungulates, proboscidia, hyracoidea, sirenia, etacea and prosimiae to associate itself with the primates. As to urine metabolism the Dalmatian is unique, and it may truly be called the dog that urinates like a man. Biochemist S. R. Benedict[1] made this observation in 1916 and, since that time, professors of biochemistry all over the country have kept a Dalmatian about for the purpose of surprising the quantitative analysis students.

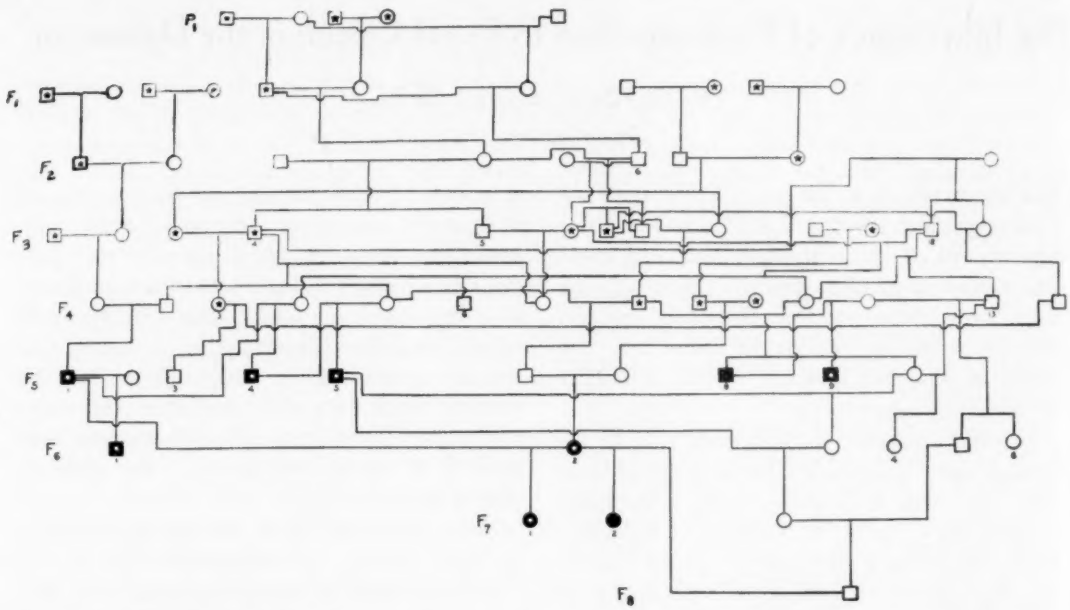
The most obvious way to account for the omission of such a chemical step was to assume the absence of the enzyme uricase, which is necessary for making this chemical transition. With this point in view, Wells[2] studied the distribution of enzymes in tissues of the Dalmatian. But he found uricase in the liver of these dogs and, hence, in this important respect the Dalmatian does not differ from other members of the species. Thus, the Dalmatian's unique capacity for excreting uric acid can not be explained by the absence of the enzyme necessary for oxidizing this substance into allantoin. The reason has been sought by many investigators and still remains a riddle.

Any departure from the normal as gross as this change in metabolism found constant for every pedigreed animal of a particular breed in the usual environment must have a hereditary basis. In fact, the breed must be almost purebred for the genes constituting the constellation of hereditary factors underlying such a unique character.

The breed must have been almost purebred for the urine character over a long time, that is, since the establishment of the foundation stock from which its modern descendants have been derived, because many Dalmatians tested in all parts of the world show the unique characteristics, and no exceptions have been reported. The Dalmatian is one of the oldest of European dog breeds. There is a quotation penned in 1807 to the effect that the Dalmatian is a dog having the "disease of drinking water" and that without plenty of water it is of little service. Since thirst is often an indication of kidney disturbances, we may suggest this statement as a proof that the Dalmatian was almost purebred for the high uric acid character at least a century and a quarter ago.

If we ask just how the high uric acid character of the Dalmatian is inherited, the reply is a long story. Onslow[3] tried to answer the question a number of years ago, but he died before the story was complete. He outcrossed a Dalmatian to a white Eng-

*From The Wistar Institute of Anatomy and Biology.



Pedigree of kidney stones in the purebred Dalmatian. (Squares = Males. Circles = Females. Stars = Bench championships. Black symbols = Autopsies. Black symbols with white dot = Kidney stones found at autopsy.)

lish Bull Terrier; then he crossed the two hybrids together to produce a second hybrid generation. The first hybrid generation was normal, and so was the second hybrid generation with the exception of one pup that developed the high uric acid of its grandparent. This work suggested that the high uric acid might represent some sort of a recessive hereditary character, but nothing more.

INHERITANCE

With the inkling from Onslow's work as a premise, we inaugurated our study. A number of questions presented themselves. Is the character of high uric acid hereditary? If hereditary, what is the mode of its inheritance? Is it a dominant requiring but one mutant member of a pair of genes to insure expression of its character, or is it a recessive in that both members of a pair of genes or allels must be mutant in order to determine "high uric acid excretion" in the individual? Is it completely dominant or completely recessive as are so many mammalian characters? Is it incompletely dominant or incompletely recessive in that hybrids are intermediate with respect to the quantity of uric acid excreted? Is it sex-linked like hemophilia, being transmitted from a male to his daughter's sons in outcrosses, because its determining gene happens to reside in the x-chromosome? Or again, does "high uric acid excretion" depend upon multiple genetic factors, as do such quantitative characters as butterfat content in the milk of cattle? If the character should turn out to be of a simple Mendelian nature involving a single pair of allels (dominant, recessive or sex-linked), is it genetically associated in any way with the "polka dot" spotting pattern characteristic of the Dalmatian? That is to say, is the gene or gene pair determining "high uric acid excretion" also responsible for producing the spotting pattern with which it has been found almost invariably in the past, just as the gene for dominant spotting in the mouse is known to produce in addition a lethal type of anemia when this gene is present in two does. If the gene for "high uric acid excretion" is not itself responsible for the Dalmatian spotting pattern, does the gene for "high

uric acid excretion" reside in the same chromosome with that for Dalmatian spotting? That is, are they genetically linked? Such a situation would simplify the explanation of why one breed of dogs should differ from all others in a character which, being unsuspected until recently, could not have been selected directly.

We collected breeding data sufficient to answer all these questions. It will be aside from our present purpose to go into all of the details of these time-consuming experiments except in a summary fashion.

1) When an anomaly is confined almost exclusively to a particular breed or family it is quite likely to be inherited.

In the pedigree of some of our crosses we have purebred Dalmatians mated to purebred Dalmatians. All the offspring of such purebred crosses have high uric acid. Hence, the character is inherited.

2) We made two outcrosses to Collies with the normal "low uric acid," *i.e.*, "allantoin excretion." A total of eleven outcross pups all had normal "low uric acid." The Dalmatian parent excreted 28 mg. for each kg. of body weight per day. All hybrids had 10 mg. per kg. per day or lower, which is the same as the Collie. Thus, there are two sharply distinguishable types with no overlapping classes. There are absolutely no intergrades.

When two of these hybrids having normal urine metabolism were crossed together, there were produced normals and abnormals in the ratio of 14:2. When the hybrids were backcrossed to Dalmatians, they produced offspring in the ratio of eight normals, seven abnormals.

When the types of spotting appearing in these two crosses were classified as to urine type, it was found that there was no association between the presence of high uric acid and the presence of Dalmatian spots. In fact, it was easy to produce a white or highly spotted dog with normal urine and equally easy to produce a black dog with the "high uric acid" characteristic of the Dalmatian. However, a curious detail has been noted, although we do not know whether it will have statistical significance in the future. In the second and third generation hybrids there have appeared some animals whose black spots have a few white hairs dispersed evenly throughout the black. These animals have been consistently normal as to their urine. We do not know what this means, if anything.

To summarize these findings we may say that "high uric acid" is inherited as an almost completely recessive, non-sexlinked unit-character, dependent for its expression upon the presence of a single pair of mendelizing genes. "High uric acid excretion" is in no way associated with the gene complex producing the polka-dot type of spotting, in combination with which it is so frequently found in the Dalmatian. Our linkage tests indicate that the genes underlying "high uric acid excretion" and the genes underlying the production of Dalmatian spotting are resident in independent chromosomes.

Kidney stones of ♂ F_2 1 in the pedigree chart.

The results of this study, which showed that eleven times the normal amount of uric acid passes through the urinary apparatus of the Dalmatian dog constantly, caused us to speculate as to what might happen to a system so overcharged with this substance. We decided that it was logical to expect an increase in the incidence of renal calculi.

We consulted the meager literature on the subject of nephrolithiasis in dogs and found it to be given as one half of one per cent. We inquired at the Royal Dick Hospital in Edinburgh and at a number of

veterinary schools only to receive the reply that the incidence in Dalmatians is no greater than it is in other dogs.

Fortunately, the records of a scientifically inclined Dalmatian fancier were placed at our disposal as well as the privilege of autopsying some of her animals when they died.

Of these interrelated animals we autopsied nine adults. In seven we found kidney stones of various sizes. In the pedigree of

Kidney stones of ♂F_6 I in the pedigree chart.

dogs closely related to the autopsied animals, we found eleven others who had urinary troubles, but these were not autopsied when they were disposed of. Thus, 18 related dogs either had or were suspected of having kidney stones.

We autopsied a Dalmatian in Scotland and one in Philadelphia. We have an autopsy record from New Orleans, and two records from our laboratory animals in Boston. All had kidney stones. We have a number of other records of suspected kidney stones in Dalmatians.

At first we thought that possibly factors in the environment, such as food and water, might have raised the incidence. When, however, we found it increased in Europe as well as in various sections of America, we were forced to the conclusion that the tendency to renal calculi is actually inherent in the breed and not dependent upon environment. If we take the one pedigree that we studied, we find that seven out of nine dogs autopsied had nephroliths. This is 77 per cent. An increase of incidence from one half of one per cent to 77 per cent is an increase in incidence of 15,000 per cent, which ought to be significant.

Of course, there is a high probable error introduced mathematically into such small numbers. Thus, the figure may be not quite as large when we have had an opportunity to examine more cases.

For this reason we should like to appeal to all veterinarians to explain our needs to their patrons who are from time to time bringing Dalmatian dogs for disposal so that they will permit the autopsy of these animals. We would appreciate receiving the kidneys and the urinary bladder tied with a string to retain its contents.

You are probably all saying to yourselves, "Just what can be done about the therapeutics of such animals?" In the first place, the nephrolithiasis seldom causes difficulty until the animal is 7 or 8 years old, and most dogs have died from other causes by that age. However, we know of running fits in Dalmatians, probably caused by the cut of a kidney stone, of deaths from unknown causes several days after violent exercise, of weakness in the back and paralysis in the hind legs probably due directly to the stones.

It is our hope to survey this breed more completely with the hope of finding animals having the normal type of low uric acid excretion from which to establish lines that are genetically free from "high uric acid" and, hence, free from the tendency to form kidney stones that this unusual hereditary type of metabolism confers upon the Dalmatian.

References

[1]Benedict, S. R.: The Harvey lectures, ser. xi (1915-16), p. 346; Jour. Lab. & Clin. Med., ii, p. 1.

[2]Wells, H. G.: Jour. Biol. Chem., xxxv (1918), p. 221.

[3]Onslow, H.: Biochem. Jour., xvii (1923), p. 334.

Hemostatic vitamin K has been produced in crystalline form. Its therapeutic merits are limited to definite bleeding due to lack of prothrombin.

Leptospirosis in Dogs

By B. J. ELANDER, B.S., D.V.M., and J. K. PERRY, D.V.M.
San Diego and San Mateo, Calif.

IT SEEMS that leptospirosis is becoming more prevalent in dogs on the West Coast. In studying the causative factors of leptospirosis, we find that it is identified with the family Spirochetaceae and the genus Leptospira. The species are icterohemorrhagiae and canicola. Although most of the research has been done on the icterohemorrhagiae species, we have found on the peninsula that the species canicola predominates.

The Leptospira organism is a long, closely-spiraled, cylindrical-looking microbe with pointed ends. The length varies from 6 to 9 μ and the width from 0.25 to 0.3 μ. It has motility by either an active rotation or serpentine motion. Staining of this organism by ordinary methods is unsuitable, but fair results may be obtained with Giemsa stain under a darkfield examination. For a positive diagnosis the best results are obtained from a blood sample which has been incubating for four hours. Then, an agglutination test is run and the results are read under a darkfield examination. The disease seems to exert its influence more strongly in the fall and winter, particularly in the winter. Recently, we have been sending our blood samples to Karl F. Meyer at the George Williams Hooper Foundation of the University of California for a laboratory report to back up our clinical diagnosis.

SYMPTOMS

In the early stages of the disease the symptoms are not very typical or easily recognized. However, in the advanced stages they can be read quite easily. The history in the majority of the cases is as follows: The dog becomes restless, the appetite is diminished or completely gone, the dog seems to want to drink water all the time, the bowels are either loose or slightly constipated, and the animal shivers excessively. If the dog is in a somewhat advanced stage of the disease when brought in, vomitus has usually been noticed, the color of which at first was yellowish, then greenish. In such cases the feces will range from a normal stool to a greenish paste or black, tarry substance. Upon examination we usually find the temperature varying from 99 to 104.5° F. It becomes subnormal as the case progresses. We may or may not find signs of an enteritis on abdominal palpation. On examining the mucous membranes of the mouth and gums, we usually notice in the early cases areas of petechiae which are becoming confluent; later these progress into larger areas and finally develop into ulcers.

If the animal lives long enough, jaundice becomes evident in the mucous membranes of the eyes and mouth. In some cases there are marked respiratory symptoms which, when accompanied by a fever, are likely to be misleading and cause one to make a diagnosis of distemper. The nose will be running or have the dried, sticky appearance of a dog with a well-advanced case of distemper. The eyes will have similar changes. There may or may not be pleurisy or pulmonary congestion. Bleeding from the mouth and anus is noted where the *L. icterohemorrhagiae* is the predominating species. Another symptom often noticed is a posterior weakness and general muscular soreness similar to rheumatic pains.

TREATMENT

Our line of treatment is more or less symptomatic and supportive. We have obtained the best results from the following: Daily injections of nicotinic acid with vitamin B_1, the dosage varying from 3 to 5 cc., and 3.75-Gm. (⅛-oz.) capsules of elixir glycerophosphate compound with a strychnine content of 1/32 gr., given three times a day. Intramuscular injections of aricyl (an arsenical tonic) are given daily in 1-cc. doses, along with any other medicinal compounds which the symptoms suggest. In

cases where there are respiratory complications we give 5 to 10 cc. of chlorna solution intravenously (a normal salt solution with a predominating magnesium ion which tends to correct the blood picture). Recently, Dr. Meyer has been producing a serum on a small scale which has proved beneficial in many cases. It is difficult, however, to obtain enough of it to get positive results. An attempt is made to get all possible nourishment into the dog. If regular diets fail, the patient is given, by the mouth, white karo syrup and orange juice. In cases where vomiting persists we give either intravenous or subcutaneous injections of saline and glucose.

PROGNOSIS

We have found the prognosis in most cases of any duration to be highly unfavorable. Cases taken in the earlier stages of the disease have about 80 per cent recovery when treated by the above method. In cases where the vomiting persists and ulcerations of the mucous membranes are marked, the mortality is nearly 100 per cent. Following are a few of the cases we have encountered.

CASE 1

Case 1 was a 4-year-old Scotty which was brought to the hospital on March 14, 1939, with a history of anorexia, constipation, and frequent vomitus of a yellow color. The temperature on admission was 103.5° F. A tentative diagnosis of gastroenteritis was made. The animal was first given an enema of a saturated solution of sodium bicarbonate. Following, intestinal antiseptics were given. A solution of karo syrup and orange juice was given as nourishment.

The temperature dropped from 103.5° on the first day to 100° on the second, 99.8° on the third and 99.4° on the fourth day. It remained at this point for a period of six or seven days. On the third day ulcerations appeared on the mucous membranes of the mouth and gums, and the vomitus changed from a yellow color to a greenish hue. At this point a diagnosis of leptospirosis was made. On the sixth day the eyes and nose were similar to those of a dog with distemper. However, this dog had had distemper two years previously. It was noticed that the ulcerations were gradually disappearing and the appetite was returning. A blood sample was taken on the sixth day and sent to the George Williams Hooper Foundation for analysis. The temperature began to climb slowly toward normal and, on March 28, 1939, the dog was discharged from the hospital.

LABORATORY REPORT

Leptospira icterohemorrhagiae—1:10 (++++), 1:30 (+++).
Leptospira canicola—1:100 (++++), 1:300 (+++), 1:1,000 (++).

CASE 2

Case 2 was a Westhighland, 3½ years old, which entered the hospital on March 26, 1939. The history was persistent vomiting of a greenish fluid, anorexia and diarrhea (blackish green). The temperature was 99.8° F. On examination petechiae were noted on the mucous membranes of the mouth and gums with the formation of small ulcers. A diagnosis was made of leptospirosis. This animal lived five days with a temperature ranging from 99.8° to 98.5°. The first two or three days we coaxed him to eat some boiled hamburger, but after that it was necessary to use intravenous injections of saline and glucose, 150 to 200 cc. (5 to 6.6 oz.). On April 1, a blood sample was taken and sent to the laboratory. The animal died the following day.

LABORATORY REPORT

Leptospira icterohemorrhagiae — 1:30 (+++).
Leptospira canicola — 1:3,000 (++++), 1:300,000 (+++).

CASE 3

A mixed breed terrier, age 5 years, entered the hospital on April 3, 1939, with a history of anorexia, vomiting and blackish green diarrhea. Leptospirosis was given as the diagnosis. Many large ulcers were seen on the mucous membranes of the mouth and gums. The temperature on admission was 99° F., but this dropped steadily until, at the time of death, it stood at 97.6°. Our supportive treatment and twice-daily injections of saline and glucose kept him alive

for three days. A blood sample was taken and sent for analysis.

LABORATORY REPORT

Leptospira icterohemorrhagiae — 1:10 (++++).
Leptospira canicola—1:3,000 (++++), 1:10,000 (+).

CASE 4

A registered Pomeranian bitch, 1½ years old, was brought to the hospital on March 27, 1939, with a history of posterior paralysis, persistent vomiting, anorexia, and a hard, black, tarry stool. On examination typical ulcers were found in the mouth. A diagnosis of leptospirosis was made. The temperature was 103° F. on admittance. It dropped to 99° and then returned to 100° for the last three days that the dog lived. A little food was taken by the animal for the first three days, but then it became necessary to resort to saline and glucose injections. A blood sample was taken on April 3. Death occurred the following day.

LABORATORY REPORT

Leptospira icterohemorrhagiae — 1:100 (+++).
Leptospira canicola—1:10,000 (++++).

The following two cases are inserted because of the fact that the laboratory reports on both cases were negative although we feel confident that our clinical diagnosis was correct.

CASE 5

A Wirehaired Terrier, 4 years old, entered the hospital on March 4, 1939. This animal was imported from England at a cost of $1,500. A history of constant vomiting, rapid loss of weight and loose bowel movement with frequent fits was given by the owner. The temperature was 102° F. Ulcerations were observed on the mucous membranes of the mouth. A diagnosis of ulcerative stomatitis and gastritis was made at the time of admission. During the course of the next month the temperature dropped to subnormal, then returned to 102°, and finally became normal. The appetite at first was good but later diminished and finally ceased altogether. The vomitus changed to a greenish color, as did the stool. The nostrils and eyes became sticky as in distemper. At this point the diagnosis was changed to leptospirosis.

The vomiting was finally overcome with anti-nausea tablets. The ulcerations persisted and the bowels became tight. During the siege of anorexia the animal was fed karo syrup and orange juice. By the middle of March a definite improvement could be seen. A sudden attack of pleuropneumonia occurred at this point. A slow period of convalescence followed. Three days before the dog was released from the hospital, a blood sample was taken and the report was negative. We believe, however, that we were correct in our diagnosis and that by the time the blood sample was taken, the Leptospirae in the blood stream were under control. The animal was released on April 12, 1939.

CASE 6

A Welsh Terrier, 3 years old, entered the hospital on April 3, 1939. A history of anorexia, extreme emaciation, and constipation was given. No vomiting had occurred. The temperature was 103° F. A diagnosis was withheld and the animal was kept for observation.

The second day at the hospital the dog ate fairly well and had a good bowel movement. The temperature dropped to 101.5°. The following day the dog seemed to be in fairly good spirit, ate well, and appeared to be recovering. However, the next day he went off feed, the temperature dropped to 99.8°, vomiting of a yellowish green fluid began and some signs of petechiation appeared. A blood sample was taken and sent to the laboratory.

The following day the animal was constipated and unable to stand on his feet. A sodium bicarbonate enema was given, also 200 cc. (6.6 oz.) of saline and glucose intravenously. That night some serum was obtained from Dr. Meyer and the dog was given 10 cc. (2.5 dr.) intravenously. For the next three days the animal improved and seemed to be on the road to recovery. On the fourth day following the injection of the serum he started to vomit a blackish green, blood-streaked discharge. Jaundice

became quite prominent throughout the mucous membranes. The animal was given 100 cc. (3.3 oz.) of blood by a direct transfusion but died the next morning.

Even though the laboratory report on the dog's blood was negative, we believe that he died of leptospirosis. A discussion of the case with the staff of the George Williams Hooper Foundation brought forth the conclusions that the agglutinins had not formed at the time the blood sample was taken.

COMMENT

Although we have presented just a few of the cases we have encountered in the past year, we hope that they are of interest to other veterinarians. We believe that within the next few years we shall gain new knowledge which will enable us to deal effectively with this dread disease, which is taking a heavy toll from dogs on the Pacific Coast.

Some Drugs Employed in Small Animal Practice

A nose-drop formula, consisting of phenol, 2 drops, tincture of iodine, 2 drops, and mineral oil, 1 oz., is useful in simple coryza, congestion of the mucous membranes and purulent nasal discharges. It eases breathing and consequently makes the patient more comfortable.

A 5 per cent potassium chlorate solution is a good mouth wash. For surgical wounds following dental surgery, ulcerations of the tongue and mouth and for canker sores, the following is recommended: Phenol, 1 part; aconite, 2; tincture of iodine, 3; and glycerin, 4 parts. Sulfanilamide, ¾ gr. per pound of body weight, together with an equal amount of sodium bicarbonate is recommended for tonsillitis, pharyngitis and laryngitis. Painting the tonsils with 4 cc. (1 dr.) each of phenol and tincture of iodine and glycerin sufficient to make 150 cc. (5 oz.) is also indicated.

In opacity of the cornea the treatment is as follows: Sterile milk in 5- to 10-cc. doses is given and repeated as indicated; a 5 per cent solution of dionin is applied twice a day and is alternated with epinephrin solution (1:1,000) which is also given two times a day. In most cases no improvement can be expected before the fifth day. Occasionally, dionin produces a temporary thickening and extrusion of the conjunctiva after the first or second application. For corneal ulcers powdered iodoform is blown into the ulcerated area. Sore eyes common to hunting dogs frequently respond to a soothing eye ointment (A. A. I.)

The most dependable treatment for vomiting, when not due to a foreign body, is a salt-water enema applied without force and at frequent intervals to establish normal peristalsis. Following the enema a bismuth protective can be given to shelter and rest the intestinal mucosa.

Occasionally, brads from cockleburs lodge in the mucous membrane lining the esophagus and produce an inflammation. A formula consisting of 0.3 Gm. (5 gr.) of bismuth subnitrate and 8 cc. (2 dr.) of mineral oil, administered at frequent intervals to keep the lining membrane coated, relieves the inflammation in a few days.

Otitis external (moist type) is always a problem with us. We irrigate the external ear with 1 to 2 quarts of salt solution. Then two or three drops of a 50 per cent solution of pure grain alcohol is instilled into the ear every day. In addition, the ears are taped to prevent flopping and to provide plenty of air. When all else fails, we apply oil of tar. It is messy but the end results are often very satisfactory. Borochlorotone is a good preparation to quiet a nervous dog suffering with ear canker or aural inflammations. *(Abstract of a paper presented by E. K. Sales, D.V.M., at the annual meeting of the Missouri Veterinary Medical Association, Columbia, Mo., January 31 to February 2, 1940.)*

Moths that flutter around a lamp are not clothes moths; clothes moths seek darkness, not light, and only their larvae damage clothing: Wool, fur, hair, feathers and mixtures of these with vegetable fabrics.

Clinical Aspects of Some Nutritional Disturbances in Small Animals*

By DWIGHT A. SMITH, D.V.M.
Ames, Iowa

SOME OF the more frequent clinical conditions encountered in small animals, with relation to nutritional disturbances, are anemia, blacktongue, eclampsia, posterior paralysis and rachitis. For the purposes of this discussion, only anemia, eclampsia and posterior paralysis will be considered.

ANEMIA

The discussion of anemia should include those anemias resulting from a decrease in the volume of blood or a decrease in the number of erythrocytes in a given volume of blood, or a decrease in the hemoglobin. This may be the result of impaired function of the bone marrow to produce erythrocytes or increased death of the erythrocytes by the liver or spleen or the loss of blood from severe hemorrhage.

Anemias, as the result of accelerated blood destruction, are associated with faulty nutrition or dietetic errors, parasitisms that include ascariasis, ancylostomiasis, coccidiosis and other debilitating parasitic diseases, including some of the external parasitisms. These, in combination with chronic infectious diseases or malignant neoplasms, cause a hemolysis of red blood cells that manifests itself as icterus or, when the destruction is too rapid and overrides the ability of the liver and spleen to carry on the evacuation of end products, a hemoglobinuria may result. Other possible causes of anemia are chemical poisonings, including lead and arsenic, chronic liver and kidney diseases, chronic gastrointestinal disturbances, lack of sunlight, hyperlactation, and leptospirosis as described by Meyer and his coworkers.[1]

Hemorrhage may result in anemia. This is usually manifested secondarily. Severe hemorrhage from a large vessel, if properly controlled, will soon be compensated for, by constriction of the blood vessels and acceleration of the heart and respiration.

Slow, uncontrolled hemorrhages over a long period (two to five days) are manifested by anemia, the conjunctiva becomes "muddy," the buccal mucosa blanched, the sclera cyanotic, and the animal shows weakness. In these cases the blood-producing organs of the body can not maintain the normal number of erythrocytes.

The treatment of anemias depends upon the primary etiological factor. After the primary etiological factor is determined and properly controlled — whether the anemia be due to hemorrhage, chemical poisoning, parasitic or infectious disease, nutrition or malignancies — the clinical treatment is primarily the same. In controlling external hemorrhage, compression bandages should be used if the hemorrhage is located where it is possible to apply such bandages. Topical application of hemostatic powders or solutions combined with compressions often is beneficial. Small doses of epinephrin or related agents are good stimulants and also constrict the smaller blood vessels, thus temporarily controlling the symptoms of anemia.

Direct blood transfusions from a healthy donor having a short clotting period give excellent results; however, an indirect transfusion of citrated blood is usually as beneficial as the direct method and is more suitable in veterinary practice. When transfusions are employed, it should be remembered that animals have blood types as do humans, and in the event of shock, epinephrin or related drugs often will save the life of the patient if used in small doses intravenously at the onset of the shock. Blood transfusions given intravenously are probably more efficient, al-

*From the veterinary division of Iowa State College; presented at the 43rd annual meeting of the Minnesota State Veterinary Medical Society, Minneapolis, Minn., January 8-9, 1940.

though it has been reported that intraperitoneal transfusions are quite satisfactory, but the effect is much slower and there is danger of adhesions among the organs of the abdominal viscera, especially if uncitrated blood is used.

The use of hypodermic hemostatic solutions is indicated and the writer has employed the agents intravenously many times without unfavorable results. Large volumes of saline solution also are indicated.

After the need of emergency treatment has subsided, blood-building materials, such as liver extract and liver concentrates, should be given. Fresh liver and an adequate amount of protein should be included in the diet. These agents aid in building and repairing lost or damaged tissue and blood. The daily administration of copper and iron concentrates also is beneficial.

ECLAMPSIA

Clinically, eclampsia ordinarily refers to a derangement of the calcium balance in relation to the other minerals and vitamins. Clinical observations indicate that the condition is a hypocalcemia with perhaps an associated vitamin D disturbance. The clinical symptoms are observed in female dogs or cats before, during or following parturition. The condition has been observed in bitches two weeks before parturition and as long as eight weeks following parturition. The symptoms vary in severity from mild convulsive attacks manifested by champing of the jaws and slight salivation that passes off in a few seconds, or severe attacks in which the patient lapses into a coma and breathes very slowly, the heart becomes slow and the reflexes obliterated. At first sight, one might mistake the condition for deep anesthesia. Animals at this stage usually die.

The average case presented for treatment is the bitch with a large litter about five to ten days after parturition. The owner indicates the dog will spend only a short time with the puppies. The bitch is nervous and any unusual noise irritates her. Frequently, the patient stumbles around in a semiconscious condition. There are periods of recovery between the sieges of nervous derangement. This continues for a day or two. Then the symptoms become more severe and are manifested as convulsions of increased length and severity. It is in this condition that the average case is presented for treatment. However, the inexperienced owner may wait until the dog lapses into a coma, death occurring unless proper medication is administered. The temperatures of these patients are irrelevant, since severe cases may be subnormal because the processes of metabolism are so impaired that they do not maintain the body temperature. In mild cases the temperature may be normal between attacks of symptoms, although during the attacks of symptoms, especially those showing convulsions, the temperature is apt to be elevated.

Parturient eclampsia must be differentiated from chill, reflex or true epilepsy, chorea, apoplexy, tonic or clonic convulsions of meningitis or encephalitis, tetanus, tonic spasms and rest periods of strychnine poisoning, uremia and many other diseases that manifest nervous symptoms. An accurate history of the patient usually will aid one to differentiate eclampsia from other conditions and diseases, although at times the picture may be clouded and, often, considerable time is required for differential diagnosis.

The clinical control of the symptoms has been facilitated by the use of calcium and associated therapy. Parenteral calcium agents used in doses of 10 to 40 cc. (2.5 dr. to 1.3 oz.) have been found efficient. These may be administered intravenously, intramuscularly or subcutaneously. If the agent is given intravenously, caution should be observed, because some patients are subject to calcium shock. Should the clinician choose to use the intravenous route, the writer suggests that half the dose be given intravenously and half subcutaneously. Intramuscular or subcutaneous administration will usually suffice except in severe cases and, here, death may occur before absorption can be accomplished.

After the initial symptoms are controlled, the problem of recurrence remains. To prevent recurrence, the patient must be given calcium and associated minerals with

vitamin D in such a form that they can be quickly and adequately assimilated. In certain cases it is advisable to include thyroid products that hasten and aid in the calcium metabolism.

Posterior Paralysis

Posterior paralysis possibly does not have a place in the consideration of nutritional disturbances, although recently it has been the observation of the writer that quite a high percentage of these cases are vitamin deficient. The indications are that the condition involves thiamin chloride, or vitamin B_1. This discussion does not include the autointoxications that accompany chronic or acute constipation or trauma that results in paralysis due to pressure on the nerves involved, and it does not apply to intoxications that result from nephritis and uremia, or choreas and paralysis resulting from distemper or other febrile or debilitating diseases. These patients usually appear to be in good physical condition; many of them are fat, have good hair coats and indicate average or above the average care. Ordinarily, the condition occurs in household pets and not in working or field dogs.

The history usually indicates that the appetite recently has been impaired and one or both rear legs have demonstrated weakness, or if only one leg is involved, the owner may think that the dog is lame in the affected leg, since the stride is shortened. An arching of the back is also observed if both legs are slowly becoming involved. As the condition progresses, paralysis occurs in both legs. The tail also becomes paralyzed and, finally, an incontinence of urine and feces occurs and the sphincter of the anus may be dilated in extreme cases. The animal remains alert and usually eats enough food to maintain itself.

The treatment of such cases includes the evacuation of the intestinal tract to remove any possibility of autointoxication, and the addition of a diuretic for one or two days is often beneficial, since it induces a greater intake of water. Such cases, however, are symptomatic and thiamin chloride used hypodermically or by the mouth usually effects the recovery. Cases that are not too severe will respond and symptoms will disappear within 48 to 72 hours. Stubborn and obstinate cases may persist and show no improvement for days. These are very discouraging and, often, one discontinues medication too soon. It is not unusual for this type of paralysis to persist for three weeks and, then, the efforts are rewarded by a slow but steady recovery.

The writer suggests the use of vitamin B_1 in 300- to 600-unit doses hypodermically, repeated once or twice daily for the first few days, then supported by oral administration of vitamins in the form of cod liver oil or other concentrated form. The addition of calcium and thyroid products also is indicated, since these agents have a specific action on nervous tissue.

During the recumbent period, bed sores must be guarded against, since soiling with urine and feces predisposes the patient to this affection.

Reference

[1]Meyer, K. F., Stewart-Anderson, B., and Eddie, B.: Canine leptospirosis in the United States. Jour. A.V.M.A., xcv (Dec. 1939), pp. 710-729.

Public Enemy No. 1

American automobiles killed 32,400 and seriously injured 1,150,000 persons in 1938. The property damage was $750,000,000. In 1937, the toll from loss of life, injury and property damage was $1,660,000,000. The steps taken to correct all this are negligible, and 17,500,000 passenger-car owners carry no accident liability insurance. In fact, automobiles are more deadly than the bombers of the war and the chances of collecting compensation are less. Neither victim nor heir gets a pension.

According to the Talmud, germs must have been known to cause disease 26 or more centuries B. C. The wearing of raw hides for clothing, spitting on the street and drinking standing water without boiling were prohibited. From these three injunctions one thinks of anthrax, tuberculosis and typhoid fever.

Calcium Gluconate Therapy (Dicalcium-Phosphogluconate)

By RUDOLPH SEIDEN,* C. E.
Kansas City, Mo.

CLINICIANS and biological chemists have known for a long time that certain neuroses of a tetanic nature are caused by disorders of metabolism which yielded to treatment that raised the calcium level of the blood and thus established their relation to calcium deficiency. Later, phosphorus and magnesium deficiency, though not clearly emphasized, were mentioned.

It appears to be true that hypocalcemia seldom exists alone but is associated to a certain degree with hypophosphatemia and hypomagnesemia.[1] This is particularly true of typical milk fever of cows, which usually appears in heavy milkers, 5 to 10 years old, after parturition. While heavy feeding is a causal factor, the mineral and watery content of the feed is also of considerable import as in plant metabolism, a phenomenon which the writer[2] has studied during the past 15 years and which is mentioned here because of its apparent similarity to tetany and milk fever from the physiological point of view.

The body of a 900-pound cow containing 68 pounds of blood and giving 3½ gallons of milk daily, eliminates every day 30 times more sugar, twelve times more calcium, twice as much phosphorus and 2½ times more magnesium than the blood contains. The intake of these four components must, therefore, be carefully balanced to insure a state of good health. Lactation does not deplete the body of other ingredients to a dangerous degree. On the other hand, when depleted of sugar, calcium, phosphorus and magnesium, disturbed metabolism (tetany, milk fever, etc.) is prone to develop.

In the form of its chloride, calcium was first used by Little and Wright[3] in 1926. It was given intravenously and sometimes supplemented by calcium lactate given by the mouth, but owing to its irritating properties, organic calcium compounds that the tissues would tolerate and assimilate better were sought. In this search, Greig of the Royal Veterinary College of Edinburgh discovered the virtues of calcium gluconate. His article,[4] entitled "Calcium Gluconate as a Specific in Milk Fever," described this almost neutral, physiologically inert, organic salt of calcium for the first time, and there is little possibility now, after all these years, of finding an organic calcium salt less irritating than the gluconate when used parenterally. The list of calcium salts which can be injected safely into the blood steam is long enough but the tissues do not tolerate them.

Though calcium gluconate proved to be of great value on account of its relatively bland action on the tissues, its chemical properties imposed difficulties. It has a low solubility—less than 4 per cent—and a higher concentration is required to insure satisfactory results. The physician prefers a 10 per cent solution and the veterinarian a 20 per cent. To obtain a nonprecipitating solution at ordinary temperatures, producers of calcium gluconate specialties employed so-called "stabilizers" to prevent precipitation of the higher concentrations. Several such agents of variable value have been found in recent years, the original one being boric acid, discovered in 1931 by de Carli,[5] an Italian chemist. To establish stability of calcium gluconate solutions, various other chemicals have been added. Among them are alkali salts, soluble calcium salts of saccharic acid, mannonic acid, lactobionic acid, boric and mannonic acid, aluminum chloride, calcium levulinate and calcium d-gluco heptonate. The calcium content often is expressed in equivalent per cent of calcium gluconate.

The objection to many of these was the effect upon neutrality (pH) of the solution which rendered them acid or alkaline and, therefore, irritant when injected. Moreover, all "stabilizers" are body-foreign substances

*Chief chemist, Haver-Glover Laboratories.

which can not be absorbed by the system. It must be remembered in this regard that each 100 cc. (3.3 oz.) of a so-called 24 per cent solution containing 20 per cent of calcium gluconate and 4 per cent "stabilizer" contains less than 1.8 Gm. (27 gr.) of calcium but 4 Gm. (60 gr.) of foreign matter to be injected into the blood stream. One must also consider that a pH approximately equal to that of normal blood (about 7) is an important property of calcium gluconate solutions.

Dicalcium-phosphogluconate (C. G. P.) starts from equimolecular amounts of calcium gluconate (20 per cent) and calcium hypophosphite (7.6 per cent). Calcium and phosphorus in this new preparation are present in the optional ratio of 2:1 (having a pH = 6.2) and should, therefore, more nearly approach the therapeutic need in milk fever, in view of the fact that the disease is not always a pure hypocalcemia. An intravenous product of the same general order is made by adding magnesium chloride and glucose (d-dextrose) in balanced amounts. While diacalcium-phosphogluconate may be used parenterally in any manner, the latter solution (C. G. P.-reinforced) is preferably employed intravenously.

References

[1]Barker, J. R.: Blood plasma changes and variations in female bovine toxemias. Vet. Rec., li (May 6, 1939).

[2]Selden, R.: Comparing Investigations About the Influence of Various External Factors, Especially upon the Ash Content in Plants (title trans.). (Paul Parey, Berlin, 1925.)

[3]Little, W. L., and Wright, N. C.: Vet. Jour., lxxii (1926), p. 185.

[4]Greig, R. G.: Calcium gluconate as a specific in milk fever. Vet. Rec., x (Feb. 8, 1930), pp. 115-120.

[5]de Carli, F.: Annali de Chemica Applicata, x (Oct. 1931), pp. 447-453.

Chloral in Ketosis

The Alston treatment for ketosis of dairy cows (=acetonemia) was praised by W. C. McConnell of Oklahoma at the Kansas State meeting in January. The dosage recommended was 3 oz. of chloral in 36 hours; this is divided into three 1-oz. doses given twelve hours apart.

Periodic Ophthalmia an Avitaminosis?

The avitaminosis theory of equine periodic ophthalmia is the one remaining to be explored. Mayhap, a known or yet unknown ingredient of one of the vitamins will some day clarify the mystery. Certainly, the pronounced effect of certain avitaminoses upon the ocular apparatus can not be overlooked in the researches on this disease.

Acute Indigestion in Cows

The passage of a three-fourth inch horse stomach tube through an Emont speculum is suggested as the first consideration in acute indigestion of cows. As a result of chewing on the speculum the patient will expel much gas. An immediate rumenotomy is recommended if the mass in the rumen is firmly packed and mixed with gas. To hasten evacuations and to control fermentation, antiferments (creolin or pine oil) in one to two pails of hot water together with a gallon of mineral oil have given good results. Tartar emetic, 12 to 15 Gm. (3 to 4 dr.), or one-half gr. of eserine salicylate is also indicated. *(W. J. Gibbons. The Cornell Veterinarian, xxx, January 1940, p. 49.)*

The Mayflower Hotel, Washington, D. C.—headquarters of the 77th annual meeting, August 26-30, 1940.

A Packer's Viewpoint on Meat Inspection*

By HARLEY D. PEET†
Chesaning, Mich.

AS A MEAT packer, I am convinced that meat inspection is needed. We were the first of the small packers in the state, outside of Detroit, to have an inspection service, and I might say that from the first, this step paid good dividends—dividends in prestige and goodwill, not only among the retail trade, who are our customers, but also among the consuming public. I believe that it had a great deal to do with our rapid growth in the past few years.

The federal bureau of animal industry and the Institute of American Meat Packers have contributed immeasurably to the education of the public in the value of meat inspection. In many ways it would probably be a good thing if the bureau could regulate and supervise the distribution of meat and meat products in intrastate business as well as interstate.

The packing business as a whole, speaking nationally, has grown to be one of the top-ranking industries. The importance of the meat-packing industry in the economic structure of the nation is indicated in the annual value of its products—$3,320,471,231. Federal inspection of meats has gone a long way to put confidence in the minds of the consuming public and has helped to make possible this large, growing industry.

The bureau has fostered many benefits through the medium of its efficient system of meat inspection and it is a loss to the industry that packers not doing an interstate business are unable to avail themselves of this important service.

What all this means nationally can be applied to our own State of Michigan. This state has been progressive and the first to grasp new opportunities. I think that this subject of meat inspection rates a great deal of consideration. To be more specific, I would like to see a state-wide meat inspection service, controlled by the state department of agriculture.

Interlinked System of State and Federal Inspection Urged

I believe that a great number of people are hesitant about eating meat. For their own health, they should eat more of it. If such people could be sold on the fact that their meat, meat products, prepared meats and sausages come from inspected packing plants and factories, I think that the consumption of meat could be increased in the State of Michigan, and our farmers and livestock raisers would be greatly benefited. Some system of state inspection in conjunction with federal inspection that would allow Michigan packers to sell federal institutions and projects would be of much benefit to both.

Meat-Inspection Regulations Not Adequate in Most Michigan Cities

At the present time we have a complicated system of city inspection throughout the state. Some good, some not so good. Each city has its own regulations and its own methods of enforcement. More cities each year are recognizing the need of meat regulations and are installing inspection services. This shows that there is a need for such an order of things, and the surprising part, to me, is that there are still many communities that do not recognize this need.

Under any system of city meat inspection we have at the present time in Michigan, the inspection covers only a postmortem examination of the animal itself and stamps are put on only the carcass or fresh cuts from that animal. I would like to see an inspection system that would go further and regulate the many manufactured items produced in every packing plant, such as

*Presented at the post-graduate short course for veterinarians, Michigan State College, East Lansing, Mich., January 22-26, 1940.
†Peet Packing Company.

sliced bacon, boiled hams, lard, packaged meats and sausage.

Milk inspection has become a well-established function in nearly every city of any size. But if the public knew the source and the condition under which some animals are slaughtered and disposed of as edible meat, they would be as much concerned about their meat supply as they are about their milk supply. During 1939 at our Chesaning plant, 8,638 lbs. of beef carcasses and 1,103 lbs. of hog carcasses were condemned and sent to the inedible tank department—nearly 10,000 lbs. besides many pounds of various meat cuts and offals. This is in addition to a considerable number of head of live stock that were brought in subject to inspection, and of which we have no records.

These figures show what is happening in only one plant. Had the animals been slaughtered without inspection, the larger part of their meat would have reached the consuming public.

One large city in our territory just this past fall turned down a proposal for meat inspection, with the announcement that they knew of no need for such regulations and that the cost would not be warranted. Yet, it is a notorious fact that dressed hogs and cattle are being peddled on the streets of that city which no inspected packing plant could handle and no other city in that part of the state with adequate meat inspection would allow to be sold.

Program of City Inspection Under State Supervision Proposed

Several things point towards the fact that now is an opportune time to consider meat inspection under some form of state supervision. I am sure that the state department of agriculture recognizes the problem and is interested in some such movement. We have at the present time a commissioner of agriculture who understands and is familiar with the meat-packing business and will be sympathetic and helpful in any forward movement of the industry. I would like to see Michigan adopt a law similar to the one operating in California, where it has been successful. Thirty slaughtering plants in Pennsylvania are operating under the official supervision of that state. They have a meat hygiene division of the state department of agriculture that is successfully trying to cope with this problem.

I know that the Institute of American Meat Packers is anxious to help establish uniform state laws all over the country. Their aid, their information and their staff of experienced men are available upon request. To a large extent their problems and those of the national packers are the same as those of the packers in Michigan. They have to contend with many state laws. We have the same problems in attempting to coöperate with the many city laws in Michigan.

Probably many of you know about the biennial attempts to change the sausage laws every time our state legislature meets. It is going to be necessary to go into all this again at the next session, as the present sausage laws are inadequate. There has never been an attempt to control the manufacture of sausage at the source of production. All manufacturers pay a considerable license fee at the present time, yet in a large part the control and supervision is maintained through the retail outlets. Meat-inspection laws and sausage regulations are so similar that it would be possible to combine them under one head and have a uniform law regulating one industry. The retailing of sausage and its control could be handled through the general food and drug laws. We have many other acts governing some branch of the industry, such as the livestock dealers and brokers licensing act, that could be incorporated in such a law and make it much easier for the department of agriculture to enforce.

Of course, in any discussion of new laws and their enforcement, the question of how they can be financed always is important. I believe it is possible, as far as meat inspection is concerned, to finance such a movement without any added expense to the state. Every sausage maker in Michigan

and every packing plant outside of Detroit that offers inspected meats to the trade is already paying out a lot of money for inspection. Meat inspection, to start with at least, could be made optional with each packer. If he would rather be regulated by whatever city inspection laws he is concerned with, he could do so, but I believe there are enough packers in Michigan who would be glad to come under some uniform state-inspection regulation with proper enforcement, that they would be glad to pay for such a service, and that a state-inspection law could be financed from such sources. A system of fees based upon the number of animals slaughtered could be established, thus making it uniform for each packer. These fees would be used by the department of agriculture to defray the expenses of a registered veterinarian at each plant, the inspector to report and be responsible only to some department head at Lansing. In plants too small to carry a full-time inspector, a system of staggered operations could be worked out whereby one man could handle more than one plant.

Any state-wide inspection law would naturally meet with some opposition, as many people would not understand it and many communities would not wish at first to come under such regulations. There would have to be considerable education and pioneering. The need and advantage would have to be explained and brought out in the open. The veterinary profession and the packers understand the problems of each other and, working together, could make much progress in such a movement.

That is why I think that such a new movement should be made optional at first, optional both on the part of the meat packer and on the part of the different communities. Certainly, enough packers would be interested from the start to make it worth while to set up the machinery for state supervision. If some packers did not feel the need of offering their trade inspected meats, they could operate as they have been doing.

Each town or city could elect, or not, to require state-inspected meats and meat products. Those cities that are already governed by their own inspection laws and are geared to enforce them could continue if they wished. In the thinly inhabited sections of our state, if these regulations are not practical, the optional feature of the law could work no hardship on them. It perhaps seems wrong to talk of a law that is optional for those concerned, but we do have such laws in other situations, and if we attempt to make a mandatory law at first, too much opposition would be encountered and a failure made before the public could realize the vast amount of good that can be accomplished.

In other words, we have to start somewhere in any course we pursue, and if the ground work could be laid on a basis of no added taxes on the part of the public and no added expense on the part of the state administration, there would be no opposition from such sources. The details could be worked out and a start could be made to cover a necessary function of good government.

Calf Pneumonia

By the use of sulfanilamide it has been possible to reduce the mortality from approximately 50 per cent to 25 per cent. Calves are given 9.8 Gm. (150 gr.) twice daily. The new drug, sulfapyridine, has likewise given good results in calf pneumonia. The calf dose is 5 Gm. (77 gr.) twice daily. Improvement is usually noted within 24 to 48 hours, *(W. J. Gibbons. The Cornell Veterinarian, xxx, January 1940, p. 48.)*

Protein Feeds for Swine

Pigs weighing 40 to 70 lbs. apparently utilize high protein rations (12, 17 and 22 per cent) more efficiently than heavier hogs, particularly those weighing over 150 lbs. Stiffness was noted in 40 to 60 per cent of the experimental animals. This was not eliminated by the use of cod liver oil or an adjustment in the mineral content of the ration. No stiffness was noted in a group of 15 pigs fed the same ration except for the addition of a manganese supplement.—*Fifty-second annual report, Pennsylvania Agricultural Experiment Station.*

Significance of Veterinary Medicine*

By D. M. CAMPBELL, D.V.S.

Chicago, Ill.

THE ART of veterinary medicine is ancient. It was already well developed when the earliest known records of civilization were made. The science of veterinary medicine is of respectable lineage. It ranks in age with the science of medicine, of agriculture and of botany, for example, and far antedates the science of aeronautics, of electricity, of nutrition and of many others that bulk large in everyday affairs. The profession of veterinary medicine, on the other hand, is rated among the youngest of the learned professions.

Few in any walk of life appreciate the dependence of man upon animals. All life upon the earth is sustained by the thin layer of soil, and man depends upon animals not only to render suitable for his use many of the crude nutrients that plants take from the soil, but also to aid the growth of the plants. With the somewhat doubtful exception of the turkey and the special case of the elephant, the history of civilization contains no record of the domestication of any animal. That is just another way of saying that until primitive tribes domesticated animals, they remained primitive. While peoples of central and southwestern Asia were developing a civilization with the help of domesticated animals, natives of Africa, Australia and North America, who domesticated no animals, remained savages.

In man's adventurous struggle from savagery, animals were an indispensable possession, providing him with food, clothing, shelter and transportation. Naturally, one of man's earliest and most important obligations was maintaining the health and improving the physical make-up of his domesticated animals, which, in large measure, provided his livelihood. Thus the art of veterinary medicine developed simultaneously with civilization itself. The oldest laws in the world, those of Hammurabi, recognized veterinary practice as a distinct art and imposed rigid regulations for its conduct. Livestock sanitation was employed for the control of infectious diseases at the time of Laban the Syrian, and in the days of Moses it had reached a high degree of efficiency, as is shown by the method of handling the Fifth Plague in Egypt, which was anthrax.

This obligation to maintain the health of domestic animals has remained one of the most essential obligations of society, from the dawn of civilization right down to the present time. The great catastrophes that have overtaken tribes, nations and even whole continents through the ages have been due, in no small part, to a failure to discharge this obligation. The extent to which even profound students of medical history have overlooked the obvious relation between animal plagues, famine and human pestilence through the ages is truly remarkable. The latest of many examples of this is that excellent work of Zinsser, "Rats, Lice and History." It is largely an extended category of dire pestilences that swept across Europe in great waves, one after another, for 1,500 years, each destroying hundreds of thousands of lives. Again and again, Zinsser mentions that the pestilence was preceded by famine; but nowhere does he mention that the famines were caused by animal plagues, a matter upon which history is almost equally explicit.

Founding of Planned Veterinary Medicine Halted European Plagues

It is more than a coincidence that waves of animal plagues, famine and human pestilence, which coursed Europe throughout the Dark Ages and the Middle Ages, came to an abrupt halt with the founding of veterinary colleges and the establishment of a scientific veterinary service in those countries. No land with a good veterinary service to conserve its live stock has suffered from serious widespread famine.

*Presented at the 76th annual meeting of the American Veterinary Medical Association, Memphis, Tenn., August 28 to September 1, 1939.

Famines there have been in modern times, but in backward countries, countries without a good veterinary service—China and India, for example.

RUSSIA'S DESTRUCTION-RECONSTRUCTION PROGRAM OF VETERINARY SERVICE CITED

Following the counter revolution in Russia, the Soviet authorities destroyed their veterinary service. Then followed the most terrible famine that has occurred in 200 years. No one knows how many persons starved to death, but few estimate the number at less than 5 millions. Pestilence, of course, ensued, for here both causes of pestilence—famine and war—were operating. Of this period, Zinsser says:

> There are no words to record the dreadful sufferings of the Russian people from 1917 to 1921. Revolution, famine, cholera and dysentery helped, but we are concerned with typhus alone. It is likely that, during these years, there were no less, and probably there were more than 25 million cases of typhus in the territories controlled by the Soviet Republics, with from two and one-half to three million deaths.

Apparently, the Russian people recognized the relation between the lack of a veterinary service and the disasters that overwhelmed them, for they immediately set about to establish the most elaborate veterinary service in the world. They now have more veterinary students enrolled in their colleges than have all the other countries of the world combined.

Western nations have enjoyed an assured food supply, at least adequate to prevent widespread famine, for so long that the general public has little realization that its immunity from famines is due to the control of animal plagues. Veterinarians themselves often forget this. One hears them discuss the contributions of veterinary medicine to public health and limit them to food inspection and the control and eradication of diseases communicable from animals to man.

The contributions of veterinary medicine are just as important to public health as food is. The loss of animals from uncontrolled plagues means not only the loss of the food and raiment represented by the animals, but also the loss of the means of tilling the land and producing those grains, fruits and vegetables which man consumes without their being refined by animals. All records of famines from the dim accounts of the Dark Ages to the story of the Russian famine following the World War speak of the pitiful condition of the inhabitants reduced to subsistence upon grass and the roots of wild plants, for that is all that is available in untilled fields. Vast, uncontrolled migrations of the undernourished and starving populace in search of food, with a relaxation of all sanitary control, set the stage for pestilence, the ultimate debacle. No nation can have an assured food supply without the control of animal plagues, and there is no insurance against animal plagues other than that afforded by a skilled veterinary service.

PREVENTABLE LOSS FROM ANIMAL DISEASES LAID TO LAISSEZ-FAIRE ATTITUDE OF PUBLIC

This country has developed an excellent veterinary service for the control of animal plagues of the conflagration type, but in the matter of more insidious diseases its attitude is not unlike that toward its hazards to human health and life. To quote Surgeon General Parran:

> No community once hard hit by yellow fever or smallpox spared money or effort to save themselves from such a tragedy again. As soon as scientific knowledge was found to fight such diseases, it was put to work. The lag was short between the discovery of such knowledge and its application.
>
> In this year 1939, however, we find ourselves confronted with an entirely different situation. We have learned a good deal about disease other than the epidemics, other diseases which kill and maim and lay waste, and load a staggering burden of expense upon the community. But in spite of the fact that there is scientific knowledge about them which is just as effective as our knowledge about yellow fever, we do very much less than we did because of fear of yellow fever or similar plagues. Partly, I think, because it frightens us less when people die one at a time, separately, of many and various maladies, particularly those of which we have no fear for ourselves or our families.

The preventable loss from animal disease in this country amounts to a staggering sum annually. No one knows even approximately its size. Disease is definitely the

limiting factor in animal production and undoubtedly costs agriculture several times the amount of the government subsidies it has received. Twenty-five per cent of all the pigs farrowed are lost from other than accidental causes before they are a week old, and only about half the pigs farrowed reach marketable age. Our hog raisers board free, the year around, 5 or 6 million sows because of this loss. Hog cholera is a disease that can be rendered all but innocuous without difficulty, and perhaps it could be eradicated; yet it costs the swine industry 20 to 30 millions of dollars annually.

The loss in incubating and baby chicks is incalculable and 30 to 70 per cent of the hens that survive chick diseases are lost from disease during their first laying period. As a result, loss is more common than profit in the poultry industry. An incapacitating but preventable skeletal disease of horses is so widespread that comparatively few farms entirely escape it and farm mortgages multiply for the purchase of tractors. The dairy industry feeds, houses, cares for and milks 24 million cows for a production that half that number should easily manage, and the principal reason is disease. If it could deduct the expense of maintaining 12 million cows from its costs, that industry would be prosperous instead of in its present impoverished condition.

The category is unending; even among pet animals the situation is not improved. The 40,000 commercial breeders of dogs in this country lose 135,000 puppies annually before they are 2 weeks of age and 50 per cent of the remainder die before they are mature. Again, a staggering loss from disease that all but precludes profit in the industry.

Superimposed upon the direct loss from animal disease is the cost of 30,000 to 40,000 remedy salesmen, chicken "doctors," breeding "experts" and other harpies who fatten upon the misfortunes of animal producers, giving nothing of value for their maintenance but rosy promises and short-lived hopes. In addition, the livestock industry supports a sizeable quack-medicine enterprise. The poultry industry alone spends $15,000,000 annually for remedies that have been proved scientifically to be worthless for the conditions for which they are sold. Although the amount has not been estimated, the expenditure of the swine and dairy industries for nostrums must be vastly greater than that of the poultry industry.

A further example of the failure of the public to avail itself of veterinary service is shown in the uninspected food that it consumes. One third of the animals slaughtered in this country for meat receive no adequate inspection at the time of slaughter or during processing. It is known that this uninspected portion of our meat supply includes annually at least 300,000 animals so badly diseased that they are unfit for food, and the probabilities are that the number is far greater. Only a minor fraction of our milk and other dairy products is adequately inspected at the source of production, with the result that a great part of these products is of low quality and unwholesome. Because of lack of confidence in the purity of such products, the demand is far below what it otherwise would be.

Thus, the significance of veterinary service presents two contrasting pictures. On the one hand, it makes possible an animal production adequate to supply the enormous population of the world today, a population four times as great at it was 200 years ago, and through its control of animal plagues, it prevents widespread famine in the more enlightened nations. On the other hand, the average annual loss from animal disease is perhaps greater at the present time than ever before. This loss is particularly regrettable in view of the fact that the veterinary service, especially in this country, possesses the information and the capacity to prevent the major portion of it. It does not do it, however, and can not do it until the public is sufficiently informed to rely upon scientific veterinary medicine for animal-disease prevention. The remedy requires a vast program of public education in the matter, particularly education of the agricultural industry. That would seem a logical task for the federal and state agricultural extension service. It has not performed it and seems little inclined to do so.

EDITORIAL

Reading maketh a full man, conference a ready man, and writing an exact man.—Bacon.

Looking into the Future

ORGANIZED veterinary medicine is integrating into a more solid unit. Loose ends are being spliced together as in other societies of the arts and sciences exploiting a nation-wide jurisdiction. It is now a going federation of the state associations of the United States and of the three principal ones of the Dominion of Canada, with the prospect that these components will seize the opportunity of coöperating in the same general way with the local organizations within their respective states.

The national medical and dental associations are founded upon the county societies and these are the components of the state society. Allegorically stated, the county is the foundation, the state the superstructure and the national the integrated whole.

In the veterinary profession, the county society, except in congested centers, could not have sufficient numbers to maintain a satisfactory working unit. But, in lieu thereof, there are existing local associations whose territory could easily be outlined to serve the same purpose as the county societies serve in medicine and dentistry.

The national association which represents the veterinary profession of the United States and Canada is looking ahead to the day when all of its membership will come through the state associations which, in turn, would recruit their members from the locals.

THE IDEA NOT NEW

The plan is not new in the veterinary circle. It was recommended 50 years ago by President Huidekoper, at the Washington session of '91, and by several presidents since that time. But, because some of the states had no organization and those of other states were weak, the advocates of the plan had to wait for developments.

FEDERATION UPPERMOST IN MIND

Although uppermost in mind all of the while in the inner councils, no definite step to put the plan into operation could be taken wisely until the second decade of the century (1916), when a new constitution looking forward to federating the state associations was adopted. North America was divided into five districts (later into ten) each to be represented on an executive board by a member chosen within his district by postal ballot.

Thus, during the past 24 years, the veterinarians of North America have had the opportunity to take charge of the executive department of the Association.

Up to that time the only threads that kept the Association "national" were its name and the resident state secretaries, whose duties were never sharply defined because a federation of state associations was always considered the main objective—the step of the near future. It was not until the third decade of the century (1935), however, when a house of representatives was formed, that this objective was attained. Organized veterinary medicine, then as now, was still in the making.

While these details belong more to the history of the A.V.M.A. than to this theme, it seems necessary to point out steps of this genre to refresh the mind on matters easily forgotten or still unweighed.

FUNCTION OF HOUSE NOT IMMEDIATELY REALIZED

The House of Representatives met for the first time at the Columbus, Ohio, ses-

sion in 1936. Lacking edification as to the responsibilities with which they had been charged, the state associations did not immediately realize the magnitude of their undertaking. The idea was new and unknown and had not impressed the members of the state associations. The House is but four years old. Canadian associations elected their first delegates to it this year. However, the keen interest shown in its affairs by the present members, but four years after its first meeting, is one of the marvels of organized veterinary medicine in North America. That the House will assert its rights and exercise its authority in the future is now self-evident. The members stand ready to guard the interests of the membership in their respective domains.

The Next Step

With the Executive Board and the House of Representatives chosen in strictly democratic fashion—devoid of lobby or pressure—recruiting memberships in the state organizations and the national association through the local ones is the next aim. In fact, the main object of this editorial is to submit this far-reaching proposal to the veterinarians of North America for study and early action.

When recruiting memberships through local organizations becomes a fact, the application of veterinary science will take on a quasi-public rôle, easily hooked up to municipal, regional, state and national veterinary work. It will become the sole unit to which the public services can go for aid, influence, and coöperation. As the medical societies came to the aid of the federal social security program, under the Wagner-Doughton-Lewis bill (1935), so would the veterinary societies come to the rescue of those concerned in the health of animals and the wholesomeness of animal products.

It is not dreaming to envisage a complete veterinary service which, through local, state and federal aid, will insure scientific attention to every animal-disease problem and every sick animal in the whole country, as the medical organizations are attempting to do for mankind. The country will not for long tolerate the waste of such an indispensable possession as domestic animals through the menace of unscientific practices. Organized veterinary medicine must plan to subsidize the indigent and to discipline the negligent for wasting property of national import or exposing the property of others to the hazards of their neglect.

But, the veterinary profession must first be organized and well informed as to its proper place before this Utopia can be achieved.

The Committee on Public Relations has already submitted plans for taking the lead in managing animal diseases and animal-disease problems, but it has not insisted that much can be accomplished in that direction until the profession fortifies itself by more attention to organization.

A look into the future, based solely by events governed by the national association, reveals a close tie-up of all associations in a unit to which all matters of animal health and wholesomeness of food of animal origin will be taken forthwith by the local health officer, the state livestock sanitary service, the federal department of agriculture, the army, and the individual owners of live stock. Organized veterinary medicine can be made the place where all of these will go for sound advice and qualified personnel.

We have the choice of falling in with the development of one great and powerful and useful society of untainted record or of groping about more or less hopelessly for all time to come.

Have You Paid Your Dues?

Though comparisons with other years show that the 1940 dues have been paid more promptly than usual, there are still delinquents to whom the executive office regrets to announce that the April issue is the last one they will receive until their dues have been paid. As the Journal is now too costly to give away, this rule, ignored in former years, will have to be enforced. Moreover, there is no assurance that back numbers will be furnished, as storing large numbers of journals on the shelves for supplying delinquents is no longer justified.

Embarrassing the Profession

WITHIN recent weeks two examples where the profession has been exhibited in an embarrassing manner have been reported to the executive office. One involves a movie, the other a radio program broadcast by an eastern station. The immediate problem is how to put a stop to these practices. In the case of radio broadcasts it is important that all who hear these programs immediately call, wire or write the station and protest the act. In addition, similar protests should be made to the firm sponsoring the program and also to the particular system, if heard over a national hookup. Moreover, complete details as to the date and time of the broadcast, the station, sponsor and, in so far as possible, a word account of what was said should be sent promptly to the executive office of the Association.

In respect to movies, protests should be made to the local theatre management, the producing company, and the National Board of Review located in Hollywood, Calif. Here again, detailed information should be sent to this office in order that the Association can lodge an official protest in a constructive manner. This information should include the following: Name of the movie, the producing company, name of the theatre, date movie shown, and a word description of the scene.

It is important for organized groups to lodge similar protests. In fact, the larger the number of protests, the better. However, the job is not complete with one group protest. Individual objections are important. It is imperative that these protests be made as quickly as possible. Prompt, individual as well as collective effort is of cardinal importance.

In most instances these acts occur through a lack of intelligent appreciation of the veterinarian, which signifies that the profession has much to do in the way of public relations. This reminds us of a case in a neighboring state in which a proposal was made to reorganize the state health board. It was suggested that the various groups interested in public health should have representation on this board. It was proposed that the personnel of the board would include physicians, dentists, pharmacists, sanitary engineers and bacteriologists, but no veterinarians. When the matter of adding a veterinarian to the board was broached to the proper authority, the reply was, "We just overlooked the veterinarian." This is only one example; others appear on the horizon every day. Surely, there must be a reason for this state of affairs—and there is.

All persons who graduate from veterinary schools are designated as veterinarians. But it isn't long until they choose to call themselves animal pathologists, meat inspectors, milk inspectors, livestock sanitarians, professors, poultry specialists, and the like. They shirk the word "veterinarian," which in reality is their true name, yet they wonder why the status of the veterinary profession requires so much edification. The medley of misnomers does a splendid job of confusing the public. It is a baffling puzzle to those we seek to educate. These terms beautifully hide the fact that all are concerned with veterinary medicine.

Recently, we were greatly impressed when visiting with a well-known physician engaged in research work. During the conversation a friend of ours stopped to say, "Hello," and we in turn introduced him to our distinguished guest. Our friend asked the research man his business. He replied, "I am a physician engaged in research work." As always, his profession came first.

In our case it is apparent that until all of these monikers are integrated into the common term "veterinarian," the organizations which honor the word by retaining it and endeavoring to build up a good name for its personnel will labor at a disadvantage. This will be reciprocated in the progress of our public relations program, which is constantly at work to better conditions for these groups as well as for those who have not dropped their only just title.

The Medicine Cabinet

PRACTICALLY every home has a medicine cabinet hidden behind the looking glass in the bathroom. A glance at its contents, however, shows that the inventory would not score high in medicines. A vial of iodine (tinct.), increased in strength beyond usefulness by evaporation, and a box of aspirin tablets are hidden away among an array of cosmetics, shaving tools and half-used bottles of favorite remedies of radio origin. Useful medicines and outfits such as one needs in emergencies are notably absent. Sixty-five thousand people are badly injured in the home every year and, yet, when these accidents occur, one looks in vain through the medicine cabinet for means of giving first aid to the wounded. Thewhella and Bishop (*Your Life*, March 1940) point out that since waiting 'til morning to call the doctor and having no emergency matériel in the medicine chest costs many lives annually, naming a list of remedies every family should keep on hand is in order. They name the following as the sensible inventory for every home:

Argarol	Aspirin
Borax	Aromatic spirits of ammonia
Milk of magnesia	Eye cup
Tinct. benzoin	Medicine glass
Mineral oil	Cornstarch
Clinical thermometer	Tinct. metaphen
Paregoric	Rubbing alcohol
Suppositories	Triple bromides
Bandage	Epsom salt
Gauze	Toothpaste
Adhesive tape	

Iodine, mercuric chloride tablets, mouth washes and cold tablets are disapproved.

THE FARM MEDICINE CHEST

The naming of this list in a popular magazine suggests the compilation of a similar one for the farm stable and, to father the thought, a canvas is being made among experienced practitioners to enable the Association to recommend precisely what should and what should not be used for the emergency treatment of animals.

Home treatment of animals is a delicate subject to broach, since the remedies used to run all the way from the deadliest of disease-producing viruses, useless serums, dangerous anthelmintics, poisonous medicines and drastic purgatives to all sorts of worthless nostrums. Making up a list of useful remedies every farmer should keep on hand is, therefore, a worthy undertaking advantageous to all concerned, including the suffering animals which, unfortunately, have often been "doctored" beyond redemption with improper remedies when professional aid arrives.

The Finnish Veterinary Service

DONALD DAY, Finland correspondent for the *Chicago Tribune*, makes the first page with his description of Veterinary Hospital No. 1 of the Finnish troops contesting the Russian invasion. He describes a clean, heated stable containing 200 patients, horse nurses and modern appointments, including the individual watering troughs of well-managed wartime hospitals for transport animals, not to mention a classical skin transplantation performed by the veterinary officer on a shell-wounded horse. A placard over each stall tells the horse's ailment, its temperature and treatment, "just like human patients have in a hospital." The horse hospital is declared to be even more comfortable than some of the field hospitals for soldiers visited by the reporter.

"In spite of mechanized warfare," says this correspondent, "the horse is helping Finland in many ways . . . animals keep supplies flowing steadily to the various fronts. . . . The Finnish horse was helping to write another proud chapter in Finnish history, the veterinary officer in charge declared."

This report corroborates previous information received about how the animal transportation of the outnumbered Finnish troops helped to keep the mechanized Russian legions from overruning their country.

We compliment the *Chicago Tribune* for making front page news of a principle in

warfare no nation can afford to set aside in the sense of security created by playing up exclusive mechanization of troops afield. The article again reminds us of calling attention to the blunder we may be making by abolishing animal transportation to a great extent in our military set-up. In a closely contested military operation, reliable transportation, not speed, is the important factor.

Reforming the Pharmacy of the Veterinarian

THROUGH RULINGS of the federal bureau of animal industry the veterinary biological products of the future are removed from the blunderbus class of therapeutic agents. They must not be labeled as cures for disease but for just what they contain in microörganisms precisely in the same manner and extent that the food and drug administration, just a few years ago, disapproved the growing practice of naming specialties after the disease for which they were to be employed.

The practice of veterinary medicine was turned from a downward to an upward direction by abolishing such ungainly names as Colic Remedy, Laminitis Tablets, Azoturia Elixir, Enteritis Tablets, Fowl Cholera Solution, etc., etc., through a long list of specialties which encouraged the doctor to leave his fine college training behind to imitate the customers of the nostrum industry.

While the reform seemed harsh to the makers of these products, none seriously interested in the progress of the veterinary service would now want to step backward to the former order, which was becoming hard to distinguish from that of the stock-remedy peddlers.

"To 'Poor Union Man': If anybody should wake up, it's you. You holler because you pay $70 union dues. That's pin money in comparison to what I pay. My dues for 1939 were $238.40 and it was well worth it.—*"Electrotyper."* (*From the Chicago Daily Times, February 8, 1940.*)

Breeding Out Low Resistance to Disease

PARTICULARLY in regard to the high mortality of adult chickens in the laying house, large-scale efforts are being made to breed strains of chickens with a higher degree of livability. Otherwise told, the poultryman must develop strains that have sufficient inherited stamina to resist such diseases as leucosis, coccidiosis, coryza, fowl typhoid, variola, cholera, etc., and to accomplish this by means of the principles of genetics. "Breeding from long-lived individuals and from long-lived families of reasonably good production averages," says A. J. Hannah in *Poultry Tribune*, "in my opinion, is going to be the solution of the mortality problem."

On the other hand, Horace Dryden of California, speaking from a long experience, declares that while breeding should be based upon family averages, there is too much of a tendency to consider this (breeding for resistance) as the only method whereby mortality can be reduced. Unless progeny records are carefully studied and properly interpreted, they may be misleading, says Dryden, who further argues that high production has nothing to do with high mortality, since good producers possess the vigor and stamina which give a strong and dependable race of fowls. Ironically, this expert declares that as a means of increasing mortality, current practices are scientifically excellent. More than 80 per cent of commercially hatched chicks come from young pullets, whereas more of them should have been bred from old hens of known livability or from selected pedigreed pullets.

Our reaction to these debates is that developing racial resistance to disease in gregarious domestic animals pushed for high production and reproduction is a complex undertaking under the present knowledge of biology.

Good language is dead language. Were one to wait for words to get into the dictionary before using them, he couldn't get a job on the *Podunk Blatter.*

The Starting Shot in the Battle Against a Disease

COMMENTING editorially on a report on the "age-old laxative idea" in the treatment of the sick, the *Journal of Digestive Diseases* cites a study of the use of cathartics in the treatment of respiratory infections published in the *Military Surgeon*, in which it was shown that in 1,013 cases (493 of which were checks) recovery was delayed and convalescence prolonged among the patients receiving castor oil or magnesium sulfate. The observation prompts a popular "digest" magazine to head its abstract, "Military Surgeon Debunks Age-Old Laxative Idea," and thus cultivates the impression that such therapeutics should be discarded as an archaic practice. In veterinary medicine such an impression would be unfortunate.

Beginning the treatment of a disease by first giving a purgative is admittedly an age-old practice and it is one that has not entirely died out. The cathartic has been the starting shot of the battle against disease since the earliest days of ancient medicine, and the practice was handed down without much change through the centuries. To the old physicians, the physic drove out bad humors. Their successors spoke of its revulsive and other actions when the humoral theory of disease was discarded through the advance of medical knowledge.

With the coming of microbiology into the art of therapeutics, the cathartic was retained to expel harmful bacteria. The modernist has retained the practice within reasonable bounds to prevent the digestive tract from becoming the hotbed of destructive microbes favored by depressed digestive action and, needless to say, to keep open and working the only avenue of body nourishment. In the latter rôles, the physic of the ancient Greek, discreetly employed, is still the savior of the sick in animal medicine, since constant attention to the alimentary canal can not be ignored as a relic of antiquity. In other words, keeping the structure and function of the digestive tract *tout ensemble* is an immutable law in animal therapeutics. In this respect veterinary medicine evidently stands apart from human medicine. The veterinarian must reckon with an extraordinarily long and horizontal alimentary tube and with an unusually large mass of contents undergoing complicated processes of reduction which, if arrested or depressed, lead headlong to a tragic end. Our teachings based upon the philosophy and science of human medicine in regard to the use of cathartics are, therefore, ill-conceived and manifestly dangerous concepts to obey.

Experienced veterinarians need not be reminded that active purgation is a hazardous practice in respiratory diseases of certain species and that in this group of infections the use of irritants to the intestinal tract is harmful. It not only delays recovery and prolongs the period of convalescence but may actually fulminate grave intestinal complications. But this exception does not signify that constant effort to keep the digestive system in order —which means moving—is overlooked by any good doctor of any animal in any ailment. Hence, with modifications governed by new ken, the veterinarian must still vote for the physic of the ancient physician.

The Twelfth International Veterinary Congress Prize

AT THE OPENING of each annual session a prize is awarded to a member of the Association for distinguished services. The award is a scroll containing the citation, and cash amounting to the annual interest derived from the residue of the Twelfth International Veterinary Congress fund collected for the New York congress of 1934.

The winner is chosen by a permanent committee comprising the chief of the federal bureau of animal industry, the director of the veterinary division of the Surgeon General's office, and the chairman of the Executive Board.

Since the award was established, the winners were D. H. Udall, Ithaca, N. Y.; George H. Hart, Davis, Calif.; and John R.

Mohler, Washington, D. C. In the order named, bovine mastitis, nutrition, and general achievements were the citations. The winner for 1940 will be announced at the opening session of the Washington meeting.

The winner of a comparable award given annually by the American Medical Association is chosen in a more complicated fashion. The intention is to spread the responsibility in such a way that no partiality can be charged. The award committee chooses five nominees whose names are submitted to the board of trustees (executive board). The trustees, in turn, select three of the five as the final candidates, and these are submitted to the house of delegates (house of representatives), which then selects the winner by blind ballot.

As selecting a recipient for this annual award may become more controversial as years go by, a modification of the method of choosing the winner may be worthy of consideration.

The winner would have to be chosen at the first meeting of the House, the day prior to the opening of the general sessions, as is done by the A.M.A. Other details, such as notifying the winner, who may not be present, and inscribing his name upon the scroll, would have to be decided upon.

While nothing has occurred or is now in sight to give birth to the thought, the removal of all personality from this significant episode of our annual conventions would certainly not be opposed by anyone, least of all by those charged with the none-too-easy task of making the choice.

Code of Ethics

THE PUBLICATION of the proposed Code of Ethics for the guidance of the House of Representatives, before it is discussed at the Washington session in August, has aroused considerable interest from every section of the country. Most of the letters concern advertising in telephone directories, which, all seem to agree, is objectionable but difficult to discontinue because of the violations by veterinarians who have no intention to follow the straight and narrow path of approved professional conduct.

Another complaint pointed out is enrolling graduates at the college door and risking the admission of members now and then who are bent upon pursuing an unethical career. This should not become a grave danger among new graduates, who were taught at college the fundamental principles of medical ethics.

Perhaps the strongest complaint of all was that received with an application for membership which reads, "I have hesitated to become a member of the Association because in our neck of the woods a membership seems to be a license to violate good ethics. Practically all of your members about here are advertisers, and in other ways show that the Association pays no attention to unethical conduct."

"The Man Is Mad"

WHEN Fulton was tinkering with a steam-driven ship on the Seine along with a French engineer (1806), the experts of Napoleon said, "The man is mad," and in 1905, when an English engineer submitted specifications for land operations much like the tank of the modern army, an expert scrawled across them, "The man is mad." In 1917, when the plans were exhumed from the dusty files and put to work, the general staff of Germany laughed at the funny notion. Came 1940 and the raiding of Poland and Finland with tanks. Stevenson, Morse, Marconi, Edison, Whitney, Howe, and many others were "mad" to experts no one remembers.

Pasteur was "mad" when he propounded his theory of fermentation and its relation to pathology. Paul Erlich was "mad" when he predicted the coming of sulfanilamide, that is, a chemical killer of germs in the living body. And who were denounced as madder men than Jenner and Lister and Morton. The trouble with the expert is that he doesn't reckon with the genius.

Reflections of the Memphis Session

HORSE AND MULE ASSOCIATION OF AMERICA

T. A. Sigler of Indiana, who is the Association's official representative in the councils of the Horse and Mule Association of America, showed that while great inroads have been made in the horse-breeding industry, the horse and mule remain the most valuable animal units on American farms, and the industry as a whole is still the richest on the list of farm-bred animals.

In its war against harmful propaganda intended to diminish the use of horses, the H.M.A.A. circulates a large amount of literature to farmers. Nearly 30,000 such letters were circulated on encephalomyelitis alone. They pointed out that the low mortality of 2½ to 3 per cent in infected districts did not justify the alarm passed along on the dreadfulness of the disease. The fact that no losses occurred where timely vaccination was practiced should remove all fear of owning horses on account of these occasional outbreaks of encephalomyelitis.

At a recent public sale in Indiana, 32 draft horses brought an average of $325 a head; one bringing $600 for a mare. A California buyer paid $1,000 for a pair of black mares. Under the leadership of the H.M.A.A., the breeding and use of pleasure horses has increased by leaps and bounds, and everywhere the interest in horse shows and pulling contests is outstanding.

AMERICAN ASSOCIATION FOR THE ADVANCEMENT OF SCIENCE

The 1938 meeting was held at Richmond, Va., December 27-31. W. B. Cannon, professor of physiology at Harvard University, was elected president. He is the first physiologist to fill that office.

The Association's award of $1,000 for the year went to Norman R. F. Maier of the University of Michigan for his work on mental diseases. There were no contributions in the section on medical sciences that were of particular interest to veterinarians. However, the meeting of 1939, held at Milwaukee on June 19-24, contained a symposium on sulfanilamide and its related compounds and one on the standardization of vitamins, which are of interest in any branch of medicine. Ward Giltner of Michigan State College has been the A.V.M.A. delegate for many years.

NATIONAL RESEARCH COUNCIL

H. E. Biester, the Association's representative to the National Research Council, complimented the late Maurice C. Hall for having established a relationship between that council and the Association. Means of obtaining additional information on the incidence of trichinosis in swine under controlled conditions was pointed out. The interest in the rabies problem was shown by the suggestion that a symposium on rabies be sponsored by the Council. The committee on medical problems common to animals and man, dealing with trichinosis, rabies and listerellosis, offered only a progress report.

State University Students and College Degrees

THE LARGE attendance at state universities is a marvel of the present period. To polish up for the dizzy whirl, young America of both sexes flocks to them in numbers unprecedented in the history of popular education. A report for 1939 of the attendance at state universities having the highest enrollment gives the following figures:

California	26,004
Minnesota	15,301
Illinois	13,510
Wisconsin	13,258
Ohio	13,231
New York	12,745
Michigan	12,715
Washington	10,129
Texas	9,872

Among the non-state universities, Columbia ranks first with an extraordinary attendance of 14,211. A glance at the figures for other state universities and endowed institutions conferring college degrees shows that the proportion of the total American

population seeking and obtaining higher education is unbelievable. If the hegira is but a chase for college degrees, the trend is unfortunate. On the other hand, if the motive is acquiring knowledge, may the attendance go on multiplying.

As President Kappel of Carnegie Corporation of New York said recently:

> Academic degrees may mean literally nothing. Only in the strong professions—notably, medicine, law and the older branches of engineering—can it be said that a degree today means anything. A Ph.D., much as it may imply as to scholarly knowledge, all too often reflects the absence of creative interest and capacity on the part of its possessor.

The JOURNAL'S rating of college degrees is different. Where creative interest and capacity to use it are equal, the "D degrees" are more than mere implications of knowledge possessed. They reflect the rational use of knowledge. But where original ideas and the knack of putting them to work are lacking, we can agree that a dangle of college degrees can have but little meaning.

"Fillers"

"WHY DO YOU intersperse miscellaneous articles throughout the reading material?" a member asks. The question is a reasonable one and, therefore, deserves a definite reply.

Articles do not conveniently end at the bottom of pages. With rare exceptions, there is some white space to fill up. What to do with it is the editor's job. He can do one of several things:

a) Start the next article with a single-column headline;

b) put a two-column headline for the next article at or near the middle of the page, according to just where the previous one ends;

c) leave the space blank, as some quite aristocratic periodicals do;

d) throw in a picture or two of some unrelated subject; or

e) fill in with short paragraphs, if possible, on some not-too-unrelated subjects.

The latter is the JOURNAL'S plan. It overcomes the indecent burial of an article under a single-column headline and gives all long articles the same display of their titles and their authors in double column at the top of a page. The plan also facilitates the make-up of reprints, many of which are supplied every month, and it does not lack the elegance all editors pride and many readers admire.

Criticism such as that cited above is gratifying. It shows that the JOURNAL is removed from the envelope and not only read but critically surveyed—the editor's dream.

Let's Watch Our Step

OUR GREATEST need today, as always, is to support the organizations that will assuredly maintain the good name the veterinary profession has earned in the United States and Canada through the leadership of the national association, and to vigorously oppose any movement that will cast any discredit, whatsoever, upon its good intentions. The good name of the veterinary service built up through the years is a precious gift won by intelligent guidance.

The public has no patience for seekers of special privilege. The slightest evidence that a profession like ours is bent upon earning more dollars from the people it serves is the surest way we know to bring fewer and fewer dollars and more and more humiliating reproach.

The argument that ours is more or less of a commercial pursuit, delegated only to save property, is taking a narrow view of a country's veterinary service. With this argument uppermost in mind, it is but a short step to the level of a common trade. We must "watch our step" because it is natural, though not always wise, to think solely of the family budget. In professional life, the dollar in sight is but the mirage that lures the pilgrim farther from the life-saving oasis.

Two delegates to the convention of the National Safety Council at Atlantic City were arrested and fined for speeding.

Organization of the United States Live Stock Sanitary Association, 1940-1941

President H. D. Port (left) and Secretary-Treasurer Mark Welsh.

Officers

H. D. Port, *President*, Cheyenne, Wyo.
E. A. Grossman, *1st Vice-President*, Cambridge, Mass.
I. S. McAdory, *2nd Vice-President*, Auburn, Ala.
W. H. Hendricks, *3rd Vice-President*, Salt Lake City, Utah.
Mark Welsh, *Secretary-Treasurer*, College Park, Md.

Committees

Legislation

J. L. Axby, *Chairman*, Indianapolis, Ind.
E. T. Faulder, Albany, N. Y.
J. M. Sutton, Atlanta, Ga.
T. O. Brandenburg, Bismarck, N. Dak.
J. P. Stout, Springfield, Ill.

Resolutions

W. J. Miller, *Chairman*, Topeka, Kan.
R. M. Gow, Denver, Colo.
D. M. Campbell, Chicago, Ill.
J. V. Knapp, Tallahassee, Fla.
R. S. Robinson, Pierre, S. Dak.

Rabies

H. W. Schoening, *Chairman*, Washington, D. C.
H. C. Rinehart, Springfield, Ill.
M. F. Barnes, Harrisburg, Pa.
J. C. Flynn, Kansas City, Mo.
C. F. Schlotthauer, Rochester, Minn.
L. E. Starr, Auburn, Ala.

Transmissible Diseases of Poultry

Cliff Carpenter, *Chairman*, Fort Wayne, Ind.
A. J. Durant, Columbia, Mo.
L. D. Frederick, Chicago, Ill.
H. J. Stafseth, East Lansing, Mich.
Erwin Jungherr, Storrs, Conn.
J. S. Anderson, Lincoln, Neb.

Committee to Study Requirements of an Ideal State Livestock Sanitary Service Organization

W. J. Butler, *Chairman*, Helena, Mont.
Ward Giltner, East Lansing, Mich.
Walter Wisnicky, Madison, Wis.
R. A. Hendershott, Trenton, N. J.
C. D. Stubbs, Little Rock, Ark.
H. A. Seidell, Des Moines, Iowa.
C. E. Cotton, St. Paul, Minn.
W. H. Hendricks, Salt Lake City, Utah.

Policy

R. W. Smith, *Chairman*, Concord, N. H.
John R. Mohler, Washington, D. C.
L. A. Merillat, Chicago, Ill.
Edward Records, Reno, Nev.
R. R. Dykstra, Manhattan, Kan.

Transmissible Diseases of Swine

H. C. H. Kernkamp, *Chairman*, St. Paul, Minn.

C. N. McBryde, Ames, Iowa.
Frank Breed, Lincoln, Neb.
L. Van Es, Lincoln, Neb.
F. L. Carr, Columbus, Ohio.

Parasitic Diseases

T. W. M. Cameron, *Chairman*, Ottawa, Ont.
Benjamin Schwartz, Washington, D. C.
E. A. Benbrook, Ames, Iowa.
I. S. McAdory, Auburn, Ala.
J. R. Ackert, Manhattan, Kan.
D. H. Ricks, Oklahoma City, Okla.

Miscellaneous Transmissible Diseases

Adolph Eichhorn, *Chairman*, Beltsville, Md.
A. E. Cameron, Ottawa, Ont.
L. M. Hurt, Los Angeles, Calif.
Hadleigh Marsh, Bozeman, Mont.
D. E. Westmorland, Frankfort, Ky.
A. C. Topmiller, Nashville, Tenn.

Meat and Milk Hygiene

A. F. Schalk, *Chairman*, Columbus, Ohio.
J. S. Koen, Storm Lake, Iowa.
T. O. Booth, Fort Worth, Texas.
E. S. Brashier, Jackson, Miss.
R. A. Culbert, St. Louis, Mo.

Revision of Constitution and By-Laws

H. E. Curry, *Chairman*, Jefferson City, Mo.
William Moore, Raleigh, N. Car.
C. D. Stubbs, Little Rock, Ark.
F. L. Schneider, Albuquerque, N. Mex.
Mark Welsh, College Park, Md.

Unification of Laws and Regulations

W. H. Hendricks, *Chairman*, Salt Lake City, Utah.
A. W. Miller, Washington, D. C.
Samuel Guard, Spencer, Ind.
W. H. Lytle, Salem, Ore.
T. B. Jones, Phoenix, Ariz.

Bang's Disease

H. C. Givens, *Chairman*, Richmond, Va.
V. S. Larson, Madison, Wis.
A. E. Wight, Washington, D. C.
C. M. Haring, Berkeley, Calif.
C. P. Bishop, Harrisburg, Pa.
W. J. Butler, Helena, Mont.
A. J. Glover, Fort Atkinson, Wis.
J. G. Hardenbergh, Plainsboro, N. J.

Tuberculosis

R. A. Hendershott, *Chairman*, Trenton, N. J.
H. M. O'Rear, Washington, D. C.
W. A. Hagan, Ithaca, N. Y.
W. H. Feldman, Rochester, Minn.
C. U. Duckworth, Sacramento, Calif.
H. A. Seidell, Des Moines, Iowa.

Why Not Castrate Them?

IT IS ADMITTED without argument that the control of rabies depends largely upon eliminating the stray dog or, more particularly, the half stray—the dog that has a home but is allowed the freedom of the streets. To handle the stray-dog problem cities maintain dog pounds and dog catchers and try in vain to keep dogs within their proper domicile. As a rule, dog pounds are wretched places cruelly managed. The situation is grotesque, comparable to trying to raise the other species of domestic animals in the same loose way dogs are allowed to be raised.

A substantial registration fee for all dogs worth keeping for breeding purposes and the compulsory castration of all others would be a step in the right direction. The step would not be cruel and it would take care of the sheep-killers, the half-starved mongrels and the maverick vectors of rabies virus. And, who can intelligently deny that a castrate is not the peer of the entire male? Moreover, the guardians of children's morality will have to admit that the entire male dog is often obscene in the presence of mixed company. When the raising of dogs is subjected to the same discipline that improves breeds in other species and controls reproduction of undesirables, it will have become an orderly enterprise that will not have to call rabies vaccination a racket.

During the past eight years 4,549 alien physicians have located in the United States. Although there has not been a comparable hegira of veterinarians, inquiries received from abroad indicate that quite a few are interested in the prospect this country has to offer them.

Farmers get 3 cents a quart for milk that costs the city consumer 16 cents. The increment is blamed on the big dairy companies and the labor unions, but thoughtful folks know that there is a lot of service between the farm and the clean bottle of milk that comes to the back door at the same moment every morning.

WITH THE EDITORS

THE LEADING article this month, contributed by H. Schmidt of Texas, represents an exhaustive study of calcium and phosphorus in relation to the nutrition of horses and cattle. The plain language of feeds and their influence on calcium and phosphorus metabolism is a feature of the paper. It contains much information that the large animal practitioner can use to good advantage.

» » »

M. A. Stewart's article, "Ovine Thelaziasis," tells about finding *Thelazia californiensis* in the eyes of sheep. This particular nematode, which was discussed in detail in the December 1938 issue of the JOURNAL, is regarded as being the only known indigenous species of the group attacking domesticated animals. The life cycle is not known. Until now it has been supposed that the dog is the natural host, but finding the worms in abundance in sheep and deer and not in dogs closely associated with infected sheep seems to indicate that the latter is in reality the host. Finding these worms in the eyes of sheep suffering with keratitis suggests that veterinarians in the western part of the country should be on the watch for this parasite. Stewart points out that horses and cattle might possibly become infected with *T. californiensis*. His reference to Oberhansley's finding the worm in California deer is ably supplemented by a report of that finding on page 542.

» » »

"Inoculating material suspected of containing equine encephalomyelitis virus intracerebrally appears to be the most dependable," says Carl F. Schlotthauer. An interesting sidelight to this work is the observation that the encephalitis provoked by the virus remains fixed at the point of inoculation for about 72 hours, or until symptoms begin to manifest themselves. Schlotthauer also shows that puncturing the brain with the inoculating needle is not injurious to that organ.

» » »

The article by the late C. P. Fitch and A. G. Karlson was inspired by the work of Sarnowiec, who reported that a filtrable form of *Brucella abortus* is elaborated when the organism is grown in the presence of tuberculin. This the Minnesota workers were unable to confirm.

» » »

Realizing that the control of Bang's disease in herds of range cattle presents problems vastly different from the average dairy herd, Marsh and Welch have laid down a plan (pp. 473-477) which is designed for range conditions. Their results are impressive.

» » »

Clyde E. Keeler will stimulate the reader with his almost amazing story concerning renal calculi in the Dalmatian. Most mammals excrete a large part of the waste products of renal metabolism in the form of allantoin; but not the Dalmatian, who is like man and his close tree-climbing relatives in that the metabolic products are excreted largely as uric acid. In Keeler's words, "The Dalmatian urinates like man." He shows that this high uric acid excretion is a matter of inheritance. Moreover, this aberration is undoubtedly responsible for the high percentage of renal calculi in this breed, as it is shown that eleven times the normal amount of uric acid passes through the urinary tract of the Dalmatian.

» » »

In all probability we shall hear much concerning canine leptospirosis in the future. For that reason, those engaged in small animal practice will profit by reading the article by Elander and Perry on the subject. It is written from the practitioner's point of view.

Drs. Crawford and Frank, in their article on tuberculous synovitis, tell what happens when pure cultures of the avian tuberculosis organism isolated from swine are inoculated intravenously into young bulls.

» » »

Harley D. Peet, a prominent Michigan meat packer, advocates state-wide meat inspection to include the small packers who do not come under the jurisdiction of the federal bureau of animal industry. Mr. Peet would have the project administered by the state, with the B.A.I. coöperating. A competent meat-inspection system encourages the consumption of meat, he advises.

» » »

According to John F. Christensen, the administration of a solution of copper sulfate and ferric sulfate reduces the output of coccidial oöcysts. This piece of work should stimulate additional investigation, as it may be the means of controlling coccidiosis in feeder lambs.

» » »

Sampson and Boley report that vigorous exercise increases the blood sugar of ewes materially, all of which may mean that when dogs chase sheep, the latter do not die of fright but rather from diabetes. Curiously enough, exercise has little effect on the ketones or blood calcium. Fasting causes a reduction in the blood sugar and but little influence on the calcium and ketones of the blood. The character of the rations appears to have a marked influence on the sugar content of the blood.

» » »

W. C. Glenney, in his article, "So-Called Shipping Fever in Dairy Cattle," states that a high percentage of the cases in his practice are due to a streptococcic infection. Sulfanilamide and large doses of pulmonary mixed bacterin (bovine) are recommended as worth while therapeutic measures.

» » »

Associate Editor Mohler's article tells of the introduction of the Nonius horse and the Puli dog from Hungary into the United States by the federal department of agriculture. Dr. Mohler has prepared this article for the purpose of answering the large number of requests he has received from veterinarians who are interested in gaining specific information concerning these animals.

» » »

"Diseases of the Digestive System in Bovines," by M. G. Fincher of Cornell University, will please the practitioner. The symptoms, diagnosis, prognosis, treatment, etc., are discussed under the general heading of indigestion, which more or less includes all digestive disturbances.

» » »

A feature of Dwight A. Smith's paper is his description of posterior paralysis in dogs associated with vitamin B_1 deficiency. He suggests 300- to 600-unit doses administered hypodermically once or twice daily.

» » »

Hurt and McCulloch present a problem that would perplex almost any veterinarian doing pullorum-testing work. Here, a high percentage of a flock of chickens reacted to the agglutination test but a bacteriological examination of the tissues of these so-called positive birds was negative in respect to *Salmonella pullorum*. However, a Gram-positive coccus was recovered consistently. A high mortality was also observed in chicks hatched from eggs obtained from these positive birds and, again, the Gram-positive coccus was recovered. Omphalitis was consistently observed in the chicks which succumbed. Preliminary tests indicate that this coccus is not pathogenic for adult fowls or for baby chicks. The so-called positive birds did not react as consistently to the whole-blood test as to the rapid plate test.

» » »

B. M. Lyon's article, "Factors Limiting Swine Production," gives a splendid perspective of the swine-disease situation. The reader will gain convincing information that he can use every day.

CLINICAL DATA

Cecitis in a Dog Marked by Convulsions*

By C. L. BLAKELY, V.M.D.

Boston, Mass.

IT IS RELATIVELY common in a busy small animal practice to be called upon to treat dogs suffering from convulsions. It is doubtful if there is any symptom which causes our clients more concern and demands more diagnostic acumen on our part than the occurrence of convulsions in our patients. Clinical experience, augmented by a study of past and contemporary literature, supports the belief that convulsive seizures are almost without exception a symptom and may rarely be considered as a clinical entity.

It has been our experience all too often that when we are called upon to make a diagnosis in these cases, we usually find a number of possible causes. When these are all corrected at once, and the dog stops having convulsions, we are left vaguely wondering just what was responsible for the symptoms; and occasionally, the thought creeps into our minds that perhaps the animal would have recovered regardless of what was done. This is particularly apt to occur when another dog, which has never had a sick day in its life, is seen exposed to the same errors. Once in a "blue moon" we see a case of which we are honestly able to say, "This dog had convulsions, this was the cause, it was removed, and there has been no recurrence of symptoms to date." When such an event occurs, we think that it is worthy of being reported, and it is one such that we wish to place on record.

Our patient is a smooth-haired Fox Terrier male, 3½ years old at the time we first saw him, and weighing about 13.6 kg. (30 lbs.), a normal weight for this individual. He was first seen at our clinic on June 6, 1938. For several months past he had been having convulsions at rather frequent intervals, not daily but once or twice every two weeks. Such attacks would appear without warning, last for several minutes and then subside, leaving the dog slightly weakened and rather nervous, but otherwise apparently normal. These attacks were typical convulsions characterized by generalized muscular spasms and rigidity, and seeming unconsciousness. There was no evidence of hysteria, or so-called fright fits.

The owner could suggest no possible cause, and careful inquiry into the feeding and management produced no clue except a tendency to eat foreign material, such as grass, stones, dirt, etc. We, of course, immediately were inclined to treat this as the exciting factor, inasmuch as other phases of his clinical examination were essentially negative, except the fecal, which showed a moderate trichuriasis. Since this dog was in excellent physical condition and was showing no irregularity in his stools, we did not consider this infestation as being of any significance in the production of the convulsions.

The patient was hospitalized for five days, during which he seemed perfectly normal. Routine blood counts (not differential) were normal, as was the urine. The serum calcium was 12.4 mg. per 100 cc.

The dog was sent home with a commercial santonin preparation to be given for the whipworms, and rigid instructions were given the owners to do all in their power to prevent the dog from scavenging or

*From the Angell Memorial Animal Hospital.

eating foreign material, since we were convinced that this was the cause of the attacks.

At this point we must explain that the dog was not brought to the hospital by the owner but, rather, by a friend of hers who had formed a deep attachment for the dog. This friend, on learning of the dog's having an attack, would drive to Connecticut and bring the dog back to Boston, a total trip of about 200 miles. As will be seen later, this extreme fondness for the dog made possible an accurate diagnosis.

From the first visit in June, to October 12, 1938, the dog was seen by us five different times, and on one occasion was hospitalized for eight days. He had numerous convulsions during this time, but never at the hospital—supporting our erroneous belief that the seizures were dietary in origin. Throughout this period the feces remained positive for whipworm ova in spite of our treatments of increasing intensity with the same santonin preparation.

On October 12, the dog had another attack and was entered for a three-day period. At this time the original owner became discouraged and the friend adopted him. When the dog was discharged, the new owner informed us that if it was humanly possible to keep this dog from eating anything but what we prescribed, she was going to do it. Accordingly, she set aside one entire room in her home from which everything but the dog's bed and water pan had been removed and where he was to remain except when either she or her father had him out on a leash.

Despite these precautions he was returned on October 30, after another attack, and remained with us for eight days, during which we discovered nothing new. He was discharged, only to return in eleven days with a recurrence. On neither occasion could the owner offer any possible hint as to what might have precipitated the attack.

Needless to say, at this time the foreign-body theory had flown out the window, and we were afraid that the owners' patience would soon follow unless we could soon find a cure for our long-suffering patient.

We discussed the case with our late chief, Hugh Dailey, and he suggested that even though the dog was showing no symptoms directly referable to the whipworm infestation, the convulsions might be due to a chronic typhilitis. Frankly, we were a bit skeptical, as we see a great many dogs with whipworms showing no symptoms other than an occasional diarrhea. We were inclined to think that the dog had a brain lesion of some sort, a theory which had the support of another associate with whom we had discussed the case. By this time we were willing to grasp at straws, and as we could think of no treatment for an obscure brain disorder, we suggested to the owner that it might be in order to remove the cecum. This suggestion met with her approval and, on November 20, 1939, the dog was operated and the entire cecum was removed.

A gross examination identified the organ as being in the typically inflamed condition seen in chronic trichuriasis. The mucosa showed a thickening catarrhal type of inflammation, being slightly pitted in many spots. The distal two thirds of the organ was moderately infested with whipworms; otherwise it was of normal shape and size. No microscopic study was made.

Our patient made an uneventful recovery and was discharged on the tenth day, post-operative, with instructions to report any developments. We have received telephone reports at intervals and to date it has been 16 months since we operated on him, and there has been no recurrence. The dog has recently been under treatment for a slight eczema—but that is another story. At least, the convulsions were stopped.

COMMENT

We have reported the occurrence of convulsions as the only symptom of a chronic typhilitis due to whipworm infestation. The patient seemed perfect in every other respect. Recovery promptly followed the removal of the cecum.

We are not clear as to the relationship of the cecum to the convulsions; that is, just how a chronic inflammation in this organ can produce such a violent symptom

as convulsions and not evidence itself in any other way. One thing we have learned, however, and that is that when the elimination of the most likely causes of convulsions does not effect a cure, we should do everything in our power to remove all disease conditions, however remote the connection may appear to be.

Myelocytoma in a Dog

By ROBERT B. McCLELLAND, D.V.M.
Buffalo, N. Y.

DURING March 1938, an aged female Boston Terrier was presented at our clinic with a hard, painful swelling below and posterior to the left elbow. The animal had become increasingly uncomfortable during the three weeks previous to examination. There was no history of injury. X-ray revealed no involvement of the bone. A tentative diagnosis of sarcoma was made. The swelling continued to increase, following, to some degree, the contour of the forearm The skin was not affected. Pain gradually became periodic.

One year later, in March 1939, the animal was again examined. No pain had been evident for the past several months. The swelling had become soft and movable and now extended from the elbow to within 2 cm. (0.8 in.) of the wrist.

On January 1, 1940, the dog was destroyed because of an asthmatic condition. The owner allowed removal of the neoplasm. A soft, pink, gelatinous mass was stripped out from a space between the skin and underlying tissues. The bone, muscles and skin appeared to be unaffected. The blood supply was derived from the subcutaneous vessels.

A section of the tumor (fig. 1) shows loosely distributed cells of a myeloid character in a delicate fibrous stroma. The nuclei are eccentric, oval or bean-shaped. The cytoplasm is acidophilic and homogeneous, though in a few cells it is neutrophilic. Mitotic figures are not common. Many capillaries and ill-defined blood spaces are present.

Because there is little variation in the cell-type described, Elton[1] suggests the term "monocytoma." In this characteristic the tumor differs from the myelocytomas described in mammals by Feldman,[2] who states that the cellular constituents vary

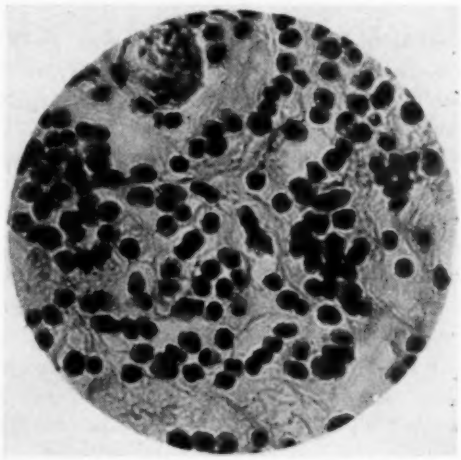


Fig. 1. Cells of myeloid character in a delicate fibrous stroma.

with the degree of differentiation attained by the type cell or myeloblast. He describes no myelocytomas in dogs.

It is unfortunate that the blood picture was not studied throughout the course of the disease and that the hematopoietic system could not have been examined at autopsy.

References

[1]Elton, N.: Personal communication.
[2]Feldman, W. H.: Neoplasms of Domesticated Animals. (W. B. Saunders Co., Philadelphia, 1932.)

Pig Anemia

Pigs from sows fed 12.5 to 70 Gm. (0.4 to 2.3 oz.) of iron during late pregnancy showed only slightly higher hemoglobin values than pigs from untreated sows.—*Excerpt from Experiment Station Record, lxxxii, February 1940, p. 235.*

On the take off the wings of the humming bird stroke at the rate of 70 per second.

California Mule Deer a Host for Nematode Eye Worms in Sequoia National Park

By FRANK R. OBERHANSLEY

Sequoia National Park, Calif.

A NUMBER of nematode eye worms (=*Thelazia californiensis*[1]) were taken from the eyes of a mature buck deer in Sequoia National Park on December 17, 1939. The deer was found lying with a broken leg a short distance southeast of the bridge at Potwisha camp. Fortunately, it proved to be a known case of "sore eyes" that had been observed at close range many times during November and December prior to the above date.

This particular species of eye worm was first described by Price[2] in 1930 from the eyes of dogs in the United States. It has since been reported from the eyes of a domestic house cat in California,[3] where it is said to be not uncommon in dogs[4] and sheep.[5] There is also one record of its occurrence in man[6] in that state.

A diseased eye condition of unknown origin in the deer of Sequoia National Park and Yosemite National Park has been described by Dixon[7] as being similar to pinkeye in some respects, and at times becoming quite widespread in areas of high deer concentration. Extreme cases were said to be accompanied by severe inflammation and accumulation of pus resulting in temporary and partial blindness from which they would usually recover in about three weeks unless taken by predators. Three similar cases and several others of milder form have been observed by the writer.

The host, from which all of the worms were taken, did not exhibit all of the above characteristics although a congested condition of the eye and apparent discomfort were plainly evident. From the time this buck was first observed until December 17, his general condition became progressively worse to the point of extreme emaciation.

When the lower eyelid was depressed, the worms were plainly visible; some were free from the eyeball and others were loosely attached in the conjunctival sac. The parasites were most numerous in the left eye, from which approximately 50 of them were removed. The most visible indication of pathology of the eye was an extreme congestion of the lymph in the conjunctival tissue of the upper and lower lids and a slight opacity of the cornea of the left eye. The amount of pus was much less than has been observed in other cases. The writer is not familiar with any previous record of this parasite in deer.

References

[1]Identification by M. A. Stewart, assistant professor of entomology, University of California, Davis, Calif.

[2]Price, E. W.: A new parasite in the eyes of dogs in the United States. Jour. Parasitol., xvii (Dec. 1930).

[3]Douglas, J. R.: The domestic house cat, a new host for *Thelazia californiensis* Price 1930 (Nematoda Thelaziidae). Proc. Helminth. Soc. Wash., vi (July 1939).

[4]*Idem.*

[5]Letter from M. A. Stewart to J. S. Dixon (Jan. 1, 1940).

[6]Kofoid, C. A., and Williams, O. L.: The nematode *Thelazia californiensis* as a parasite in the eye of man in California. Arch. Ophthal., xiii (1935), 2.

[7]Dixon, J. S.: A study of the life history and food habits of mule deer in California. California Fish and Game, xx (1934), 4.

Acetonemia Relapses

A new idea in preventing relapses following the appropriate treatment for acetonemia is the use of anterior pituitary extract. The dose is 10 cc. administered subcutaneously. This agent when used alone will cure many cases, although recovery is slower and several injections may be required. The greatest benefits of anterior pituitary extract are derived in those cases which have a tendency to relapse. Therefore, it appears best to use both dextrose and the extract. *(W. J. Gibbons. The Cornell Veterinarian, xxx, January 1940, p. 50.)*

Turkey raising is an estimated $70,000,000 a year industry.

Filariasis in Wild Swans*

By E. R. QUORTRUP, D.V.M., and A. L. HOLT, D.V.M.

Brigham, Utah

INFESTATION of Whistling swans (= *Cygnus columbianus*) with a filarial parasite of hitherto unknown identity has aroused a due amount of concern at the laboratory of the Bear River Wildlife Disease Research Station, at Brigham, Utah.

The parasite was first observed by Don R. Coburn and the senior author in February 1938. A swan found dead in the marshes displayed a moderate infestation of filaria worms although the primary cause of death seemed to be lead poisoning. A few of the parasites were found directly under the epicardium, some in coiled, others in extended positions. Other mature parasites were found imbedded deep in the myocardium. Blood smears taken after death from the abdominal cavity and stained with Wright's stain revealed numerous microfilariae. Later attempts to demonstrate the larval form of the parasite in the blood stream of living birds have been unsuccessful thus far.

About a year elapsed before another case came to our attention. Since then, incidents have not been uncommon. It is possible that earlier infestations had escaped our attention, since the condition was unknown to us and, hence, was not looked for.

Of 22 autopsies performed on Whistling swans during 1937 and 1938, only two cases showed the presence of the nematode, and both of these were in 1938. In 1939, filariae were found in ten out of 42 swans examined; this represented a frequency of occurrence of 23.8 per cent.

Even though the primary cause of death in most of the swans examined indisputably was plembism, considerable significance is attached to the presence of the filaria worms. The relative pathogenicity of the organism remains undetermined; however, apparent damage results from invasion of the cardiac wall as well as possible circulatory obstruction. No mature parasites have been observed thus far in the cardiac chambers.

Late in 1939, the nematode, which was studied by E. E. Wehr of the federal bureau of animal industry, was classified and named by him, and is now known as *Sarconema eurycerca*.

In December 1939, we received a Whistling swan for postmortem examination from Moscow, Idaho. This is the only case that has come to our attention in which all evidence points to filariasis as the primary cause of death. No lead was found in the gizzard, and chemical tests for metallic poisoning were negative. A few mature *S. eurycerca* were observed macroscopically under the epicardium. Blood smears made from the endocardium disclosed a heavy infestation of the larval form of the parasite. Examination of several Wright-stained slides revealed from one to five parasites under each low-power field. This serves to give some understanding of the enormous number of larvae present. A photomicrograph of one of the blood smears is reproduced herewith (fig. 1). Measurements were made of five microfilariae taken at random, the average size of which was found to be .26 x .001 mm. The mature parasite has been found to measure 20 to 40 mm.

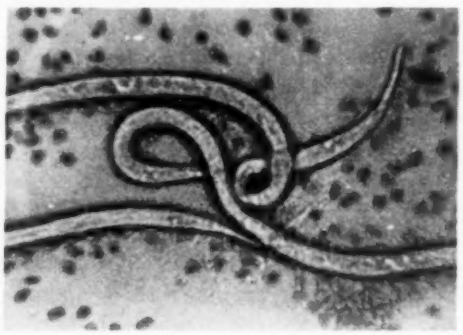


Fig. 1. Sarconema eurycerca (x 440).

*From the U. S. bureau of biological survey.

As far as is known at present, apart from academic and diagnostic value, the occurrence of this parasite has little significance, inasmuch as the stage of disease is usually too far advanced for treatment when a wild swan is so weakened that it may be captured. This is particularly true if filariasis is combined with lead poisoning. If, however, an opportunity should present itself for medicinal treatment, such an experiment will be attempted.

Carotenosis

By A. GREENFIELD, D.V.M.

Ottumwa, Iowa

VITAMIN A is important in the prevention of colds, infections, sore eyes and mouth, nutritional roup, sterility, intestinal disorders and paralysis. This vitamin may be found in carotene and is important to the cattle industry because it helps to maintain the health of the animals. An excessive amount, however, will produce a pathological condition known as carotenosis.

Carotenosis is restricted to the bovine species and involves only the liver and associated lymph glands, namely, the hepatic, posterior mediastinal, mesenteric and adrenal. The writer has been unable to find any macroscopic signs of this condition in any other organ or gland.

Carotenosis is the deposition of carotene in the liver cells. This substance changes the normal color of the liver to yellow or reddish yellow. In mild cases carotenosis resembles the color of calf liver. Where a microscope is not obtainable, the color may be fixed and brought out to a better advantage by placing a piece of the liver tissue in boiling water for five minutes. Microscopically, it has been found[1] that the protoplasm of the cells in the center of the lobule appear cloudy or granular, and there will be noted a well-marked parenchymatous degeneration with round-cell infiltration and fibrous proliferation. As the condition progresses there is a pronounced fibrous degeneration and the germinal center of the lymph glands becomes a homogeneous mass.

Upon macroscopic examination the liver is yellow to reddish yellow. White paper becomes stained with carotene when wiped across a cut surface. The borders of the liver are rounded, as if due to some irritation, and the capsule is thickened. The lobules of the liver become cirrhotic and eventually become calcified.

There is no doubt as to the identity of the substance under discussion, for the pathological division of the federal bureau of animal industry, after careful chemical analysis, has identified it as carotene. Carotenotic liver tissue was fed to experimental rats by the bureau and it was found that the material produces degenerated and necrotic areas in the livers of rats but that carotene is not deposited in the liver cells.

Carotenosis is not confined to one sex, and age is not a factor. The history obtained thus far regarding the origin of cattle with carotenosis indicates that the condition is found most frequently, if not solely, in animals from the southwestern part of the United States.

Reference

[1]Buckley, Joss, Creech, and Couch: Carotenosis of bovine livers. Jour. Agr. Res., xl (June 1930), pp. 991-1005.

Lingual Trichinosis in a Cat

By H. D. PRITCHETT, V.M.D.

Philadelphia, Pa.

AFTER A CLIENT had lost two valuable cats in one day, the writer was called to view the body of one of them for the purpose of making a diagnosis and to advise the client how to prevent further losses, as she had about 15 others.

Having been sick for several days, the cat, a male aged 5 years, would attempt to take food and water but appeared unable to swallow and, after a tedious try at deglutation, would drop the food, and then repeat the effort. The mouth was noticed to be only partly closed for about three weeks, and during the last few days of life the lower mandible appeared to be paralyzed, yet it definitely was not. Sali-

vation, of course, was profuse, stringing and constant.

The cadaver, which was obtained for autopsy after much persuasion, was in fair condition and not at all emaciated, as one would expect. The abdominal viscera were not greatly altered grossly, but on histopathological examination the liver and kidneys showed cellular infiltration. There was some degree of intestinal catarrh differentiated with cellular infiltration of the submucosa. There was a generalized stomatitis. The base, dorsum and tip of the tongue presented ulcerations containing a yellow, cheesy substance. Histopathological examination of the tongue revealed the following: Ulceration, mucosa eroded, depths of ulcers occupied by granulation tissue, encysted trichinae in muscle. There were several identical lesions on the gums. There was a cyst the size of a hickory nut located on either side of the tongue near the isthmus of the fauces, which partially occluded the esophagus and trachea by pressure and volume; these were probably ranula due to occlusion of the ducts by the stomatitis, which obviously had become chronic. Microscopic examination of these cysts revealed glandular tissue enclosed in a very loose areolar tissue.

The origin of this parasitism was not definitely established, but due to the close proximity of the client's home to a public dump, it is assumed that the infestation was obtained from rats, with which the dump was abundantly populated, as the cat in question spent much time there hunting and destroying these rodents. The original source of infestation for the rats is not known, but feeding upon the carcasses of swine dead of disease, the viscera of slaughtered swine, or discarded pork products was in this case probably the primary source of infestation. Under the circumstances, it is unlikely that the cat received the infestation from the primary sources suggested.

All laboratory examinations were made by the Pennsylvania bureau of animal industry at Harrisburg, Pa. As trichinosis was not suspected clinically, the diaphragm was not submitted.

Protein Poisoning

By C. E. BASSLER, D.V.M.

Ainsworth, Iowa

CASE 1

A herd of 30 thrifty, unvaccinated shoats, each weighing about 45.5 kg. (100 lbs.), were turned into a soy bean field on September 27, 1939. The uncut portion of the hay was fully ripe but, since it was not needed, it was allowed to stand. On September 28, several of the pigs were in a semicomatose state, and two were dead at 8:00 a.m. When the writer arrived, at about 10:00 a.m., three more were dead and ten or twelve were in more or less constant motion, running blindly or aimlessly. Later, several became prostrate and helpless. The temperatures ranged from subnormal to normal. Breathing was rapid but not labored. Diarrhea was present and the excrement was yellow.

On autopsy there was enlargement and degeneration of the kidneys. The hilus, ureters, and mucous membranes of the bladder were congested. The urine was bloody.

Treatment.—Thirty cc. of dextrose was given in the axillary space to ten of the shoats. On October 2, it was reported that all of the treated animals except one had recovered.

CASE 2

On November 18, 1939, seven healthy sows and their 6-week-old litters—a total of 59—were turned into a soy bean field where some uncut ripe hay was standing, except that which had lodged. These sows and pigs had access to the field for several days and, during that time, several pigs died. A sick pig was brought to the writer's hospital. Breathing was rapid and the animal was in a semicomatose state. The temperature was normal. As in case 1, diarrhea was present and the excrement was yellow.

Autopsy revealed the same urinary-system abnormalities that were observed in the first case.

Later that day, the writer made a call

at the farm. Several of the pigs had a yellowish diarrhea, one was in a semi-comatose state, and the balance were fairly alert and ate some. The only treatment used in this case was a vitamin-mineral powder.

Another call was made on November 24, at which time the losses were increasing and many of the animals were sick. About 15 were dead. Some of the pigs had a normal temperature, others a slight fever. The same symptoms observed on the previous call were still in evidence.

Autopsy revealed the same urinary pathology along with lesions suggestive of cholera.

Treatment.—Virus was not given but large doses of serum were administered. All died, however, within ten days. It seems certain that if the urinary complication had not been present, 25 to 30 per cent of the cholera-affected animals could have been saved. We vaccinated 150 pigs for the same owner on that day, on an adjoining farm, with serum and virus and not one animal was lost.

CASE 3

On December 4, 1939, we vaccinated and wormed 56 healthy pigs weighing about 23 kg. (50 lbs.), using 2½-gr. santonin capsules. They were being fed an abundance of skim milk and a poultry mash containing fish meal.

Two were dead and three were visibly sick when the writer called on December 21. The temperatures of the sick pigs were subnormal and a yellowish diarrhea was present.

On autopsy of two of the pigs the pelvis of the kidney, ureters and bladder were distended with bloody urine. The ureters were three times their normal size. There were no petechial hemorrhages.

The virus in this case had been used on ten other herds and the serum on 18.

Treatment.—The owner was advised to reduce the amount of skim milk 50 per cent and to eliminate the poultry mash. He was instructed to guard against giving any other kind of protein until the animals recovered. In three days improvement was reported, and no further losses occurred.

COMMENT

In all of these cases the quantity of protein consumed was not known. In case 2, the owner made an investigation after the pigs became ill. He reported that the soy beans on the ground under the uncut hay that had lodged were in poor condition, and this, in itself, without the protein factor, would cause sickness. Furthermore, these conditions also exposed the animals to cholera. Case 3 demonstrated the effect of a high protein ration, the exact quantity and quality of which were not known. Also, neither the age nor the quality of the fish meal in the poultry mash was known.

New Poultry Disease Bulletin

L. D. Bushnell and M. J. Twiehaus of Kansas State College are authors of the revised and enlarged bulletin No. 284, "Poultry Diseases, Their Prevention and Control." It consists of 125 pages, is profusely illustrated, and the subject matter is divided into 19 chapters. Copies may be obtained by addressing a request to the Agricultural Experiment Station, Kansas State College, Manhattan, Kan.

Licensing in Biological Production

The U. S. Public Health Service supervises under specified regulations the production of biological products used in human medicine but does not endorse in any way the products for which licenses are granted. The equivalent in veterinary medicine is the supervision of the U. S. bureau of animal industry over the same class of products used for animals.

The act of Congress governing the licensing of the manufacturers of biological products for human use was passed in 1902. The production is controlled by the surgeons-general of the Public Health Service, of the Army and of the Navy, but in this rôle all are under the direction of the secretary of the treasury.

SURGERY & OBSTETRICS

Treatment of Hypertrophy of the Canine Prostate Gland*

By A. A. HERMANN, D.V.S.
Denver, Colo.

HYPERTROPHY of the prostate gland in dogs can not be treated through the urethra, as is done in human surgery. The inflexible penis, curved route and small size of the urethral canal are the chief obstacles. Approaching the prostate directly through a laparotomy involves the risk of peritonitis resulting from an escape of pus when the gland is incised. There is also danger of injuring the urethra at this point.

Massage is frequently beneficial, especially in those cases which exhibit a purulent urethral discharge without the other suggestive symptoms.

In some cases rectal palpation will reveal a greatly enlarged fluctuating swelling of the gland. If punctured with an aspirating needle, a creamy pus can be drawn off. Unless swellings of this type interfere with defecation and micturition, tapping is not recommended because of the danger of peritonitis. Fatal peritonitis may occur if the fluctuating mass ruptures during the violent straining provoked by the associated constipation. When a perineal hernia results from straining, the swelling under the tail resembles the bustle on the back of a woman's dress. The hernia ordinarily contains the hypertrophied prostate, the bladder and one or two loops of the floating colon.

Ordinarily, it is impossible to reduce the hernia manually, the bladder becomes greatly distended and it is frequently impossible to pass a catheter. Relieving the bladder is best done by tapping with a sterile hypodermic needle which is inserted near the margin of the rectum. Surgical reduction of the hernia includes anchoring the bladder and colon to the abdominal wall. Even following the correction of the hypertrophied gland, it is necessary to provide for frequent micturition and defecation until a complete recovery has been made.

Surgical treatment of the gland can best be effected by castration. Obviously, it is not costly to the client, provides less risk and is convenient for the veterinarian. Three methods may be followed: Complete removal of both testicles, severance of both spermatic cords without removal of the testicles and the removal of a section of the vas deferens.

Highly satisfactory results are obtained by removing the testicles. This is done under nembutal anesthesia and through a median line incision less than 1 in. long and 1 in. anterior to the scrotum. After incising the skin and subcutaneous tissues down to the tissues of the penis, the assistant forces one testicle forward so that the enveloping membranes can be readily incised. With traction and rotation the testicle can be squeezed through the incision. The cord is then ligated with sterile catgut or crushed with an emasculator and the testicle removed. All hemorrhage must be permanently controlled or a hematoma will develop and lead to serious complications. Two Ferguson-type forceps with tongue and groove blades are applied to each cord and, in addition, the stumps are treated with an electric auto-cautery. When all hemorrhage has been controlled, the stumps are retracted and the incision is closed with four or five interrupted sutures of dermol. The area is covered with gum-

*Presented at the small animal clinic of the 76th annual meeting of the A.V.M.A., Memphis, Tenn., August 28 to September 1, 1940.

mastic dissolved in benzol and a layer of sterile cotton is then placed on the surgical wound. The sutures are removed on the fourth or fifth day. Occasionally, the patient will lick the wound and remove the sutures prematurely, but, even though this does occur, the edges will remain united and heal promptly without swelling or other complications.

A lapse of about four weeks is required for the hypertrophied gland to evacuate its pus through the urethra and shrink to normal puppy size. Precautions to avoid constipation should be continued until recovery is complete.

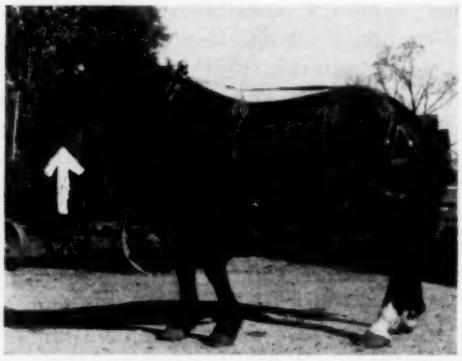
Compound Fracture of the Mandible*

By W. F. GUARD, D.V.M.
Columbus, Ohio

THE SUBJECT was a 9-year-old mare, admitted to the hospital on April 7, 1939.

HISTORY

The animal had an enlargement on the left side of the lower jaw that had persisted for over a year, with some discharge at times. The condition had grown worse when the patient was brought in for treatment. She slobbered and spit hay when eating. These latter symptoms came on suddenly two days previous.



This photograph was taken at the time that complete recovery was reported. Although there was apparently a solid union of the fracture, with the animal eating normally, some enlargement of the left side of the lower jaw still persisted, as indicated by arrow.

SYMPTOMS

There was a fistulous tract on the ventral edge of the left mandible in the region of the enlargement, ventral to the second premolar tooth. The animal was drooling saliva, and there was a foul odor from the exudate both inside the mouth and from the fistulous tract. The second premolar tooth was worn down to the gum line and the tooth above was correspondingly longer. There was considerable lateral movement between the premandible and the posterior portion of the left mandible. There was no crepitation due to the fact that the quite extensive suppurative osteomyelitis caused the two fractured ends to be separated considerably.

TREATMENT

The animal was given 45 Gm. (1.5 oz.) of chloral hydrate intravenously and the second lower left premolar tooth was removed. The fistulous opening was enlarged on the ventral border of the mandible so as to give ample drainage. The cavity between the fractured ends of bones was thoroughly cleansed and all surfaces wiped with gauze saturated with Bipp paste. A small seton was passed through the tract into the mouth, and a plug of thoroughly vaselined gauze was inserted as a false tooth in front of the third premolar. The other end of the seton was tied to an oil-soaked plug on the ventral border of the mandible.

The animal went home on the 13th, with such a pack in place, and the owner was advised to nurse the animal along on soft, palatable foods with a hope that she might recover. Two months later, June 6, the local veterinarian, Edwin R. Stockwell of Mechanicsburg, Iowa, reported a solid union. The animal was eating all kinds of feed and there was no odor, but a small fistulous tract was present. On January 1, 1940, complete recovery was reported.

*From the College of Veterinary Medicine, The Ohio State University.

Invagination of the Intestine in Ruminants

To EXPLAIN the mechanism of intestinal invagination numerous theories have been advanced. The most common and perhaps the most logical one is tumultuous peristaltic action in a segment of bowel anterior to a dilated, paralyzed or inactive segment. The one simply rolls into the other, whose caliber at the moment the accident occurs is the larger.

Another theory is that of adhesions. One spot of a bowel adheres to an adjacent bowel through the rapid action of an inflammatory process. The place thus bridled becomes the point of entrance for an invagination. Falling, running, jumping and rolling about (as in colics) are entertained as possible causes. "But," says Rehaut, "these theories do not explain the cause of the accident in the large ruminant, where the trouble occurs most frequently in the middle of the night and in stabled animals." Here the probable cause is the anatomical disposition of the viscera. As the intestinal mass rests upon the right lobe of the rumen, the intestines are subject to variable displacement depending upon the fullness of that reservoir and the posture of the animal. A full intestine at one point anterior to an inactive portion at another point under variable circumstances can, entirely independent of any other cause, lead headlong to a mechanism favorable for an invagination.

There are certain quite characteristic symptoms. A period of violent excitation with loss of control, striking the abdomen with the hind legs, turning the head to the flank while lying, and frequent defecation in small quantities are among the symptoms presented. Bloating on but the right side is a precious sign. Borborygmus is infrequent but strong. The pulse is feeble and small. There are periods of calm between crises, which may give the impression that the case is but one of mild colic. The pronounced lassitude and sorrowful appearance during the period of calm are characteristic signs during the first 24 to 36 hours. The pulse becomes stronger and faster and the painful reflexes are no longer manifested through the sympathetic. When the invaginated segment begins to mortify, the state of the patient seems satisfactory to the attendants. "She is much better," the owner says. The patient may even eat and drink moderately. The right-sided bloating continues while the left side is sunken. Auscultation reveals absolute silence on the right side and slight crepitation on the left. The animal finds relief by lying upon its left side.

Rectal exploration at this time is revealing. The seat of the invagination may be located in the form of an intestinal mass the size of an orange. It feels somewhat like the posterior aorta. By these tokens the diagnosis may be declared on the third day. Death occurs on the fifth to the seventh day.

As the location of the trouble is beyond the range of direct or indirect exploration, the diagnosis is naturally difficult, since the outward expression of the disease corresponds with that of other colics.

The author mentions surgical intervention as the only successful treatment. Under local or epidural anesthesia, a long incision (20 centimeters) is made through the right flank, the intestine is brought out, spread over a sterilized cloth, and a lateral anastomosis performed. At the time a diagnosis is made, the adhesions already prevent unraveling of the invagination. At the end of 15 days the cure is complete. [Unhappily, this optimism is not ratified by any case reports.—Ed.] (*M. le Dr. Rehaut. L'Invagination Chez les Bovidés. Recueil de Médecine Vétérinaire, cxv, February 1939, pp. 84-90.*)

The pruritus following the bite of the mosquito is caused by saliva inoculated into the sting.

In his recent revision of *Diseases of the Genital Organs of Animals*, W. L. Williams disagrees with the common belief that colostrum has the laxative and antigenic properties generally attributed to it.

Shock: Circulatory Failure

Many far-fetched but always unproved theories have been brought into the attempt to define the state of collapse associated with injury, the lesionless debacle of the vascular system following grave accidents and major surgical operations commonly known as shock. As far as the physiology of the condition is concerned, the word means nothing in particular. The vagary of the word has mislead the theorist and novice but never the capable surgeon, who always has been keenly aware that the true nature of shock is not at all baffling. To him, the collapse was easily explained as an embarrassed circulation either from the breaking down of blood propulsion or of blood distribution. The cause is always the same, namely, diminished blood volume in the large vessels, either from hemorrhage or peripheral pooling.

As told by Harvey (*Journal of Surgery, Gynecology and Obstetrics,* January 1938):

> Failure of the circulation of the blood is usually due to either a deficient propulsive force or inadequacy of the distributive apparatus. That the latter is most frequently due to a decrease of blood volume as a result of the inability of the blood vessels to retain all or some of their components, does not invalidate the significance of the term "vascular failure."

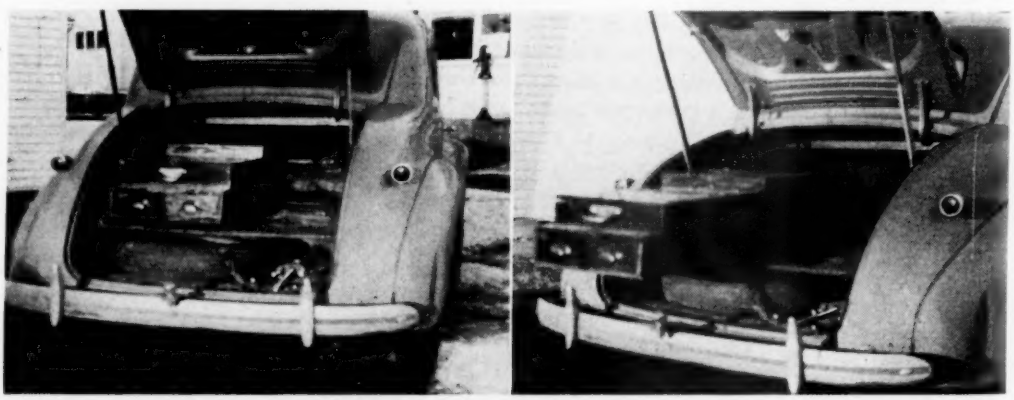
This author would abandon the word *shock* and replace it with *cardiac failure* and *vascular failure,* depending upon whether the deficiency is propulsive or distributive. The suggestion not only clarifies the subject but also gives the modern conception of what shock actually is.

Sodium Citrate in Hemorrhage

Sodium citrate is the heroic treatment for grave hemorrhage, says Jean Druillet.* It prevents augmentation of the volume of hematomata and post-operative hemorrhages. Likewise, it gives important results in the nose bleed of race horses; it not only stops the bleeding but also prevents a recurrence of the accident. By the addition of sodium citrate to the chloral solution used for general anesthesia in horses, the danger is diminished.

The therapeutic dose is 30 Gm. (1 oz.) in 100 cc. (3.3 oz.) of water for large animals (horses) and 10 cc. (2.5 dr.) of a 20 per cent solution for dogs. These doses given intravenously occasion no serious reaction.

*Revue de Médecine Vétérinaire, xci, April 1939, p. 222.



A new, handy carrier for automobiles, designed by R. M. Carter, D.V.M., of Alexis, Ill. This compartment fits a Chevrolet coupe and is 51 in. long. It has two drawers in front with a miscellaneous tray over them. Back of these drawers there is room for twelve 8-oz. prescription bottles, twelve 4-oz. prescription bottles, and twelve 4-oz. dauber-top bottles. The bacterin compartment holds six 250-cc. vials in their cartons. The next compartment holds eighteen 500-cc. screw-cap tablet bottles, and the next, two 1-gal. and one 1-qt. bottles.

CURRENT LITERATURE

ABSTRACTS

Blood Picture in Trichinosis

In experimental infections in albino rats there was no alteration of the erythrocyte count. Total leucocytes were approximately doubled after doses of 600-700 larvae but the number was practically not changed after doses of 25-50 larvae. In severe infections a neutrophilia and lymphopenia appeared followed in a few days, however, by a lymphocytosis. The eosinophile count showed no appreciable change. This was also true of basophiles and monocytes. In so far as these studies go, they indicate that the eosinophilia, so characteristic of trichina infections in man and swine, is not elicited in the rat. (*E. H. Beahm and C. M. Downs. Differential Blood Picture and Total Cell Count on Normal and Trichina Infected Albino Rats. Journal of Parasitology, xxv, 1939, pp. 405-411.*)

Experimental Beriberi in the Silver Fox

Veterinarians in Norway have occasionally seen spontaneous cases of B_1 avitaminosis in foxes, minks and ferrets on fur farms. The first symptoms are loss of appetite, weakness and depression. Later, they show disturbances of motility and tend to roll and twist their bodies. In extreme cases the animals have clonic spasms of the limbs, masticatory spasms and opisthotonos. Treatment with vitamin B_1 results in recovery. Experimental beriberi was produced in the silver fox by feeding a diet free of vitamin B_1. It consisted of casein, cornstarch, vegetable fat, autoclaved brewer's yeast, dried whale meat, salt and calcium carbonate. Vitamin C was supplied in cod liver oil and tablets.

The first symptoms of diminished appetite and weakness appeared in six to eight days. The clinical symptoms of beriberi were evident in 18 to 21 days. The foxes showed clonic spasms of the jaws and extreme salivation. This was followed by convulsions, severe opisthotonos and spasms of the legs which would last for a few seconds up to several minutes. The symptoms rapidly disappeared after treatment with vitamin B_1.

The veterinarian who attends fur animals will seldom see cases of B_1 avitaminosis showing these extreme polyneuritic symptoms, but shortly after the whelping season the young may show evidence of vitamin B_1 deficiency by inappetence and depression. During the later stages of gestation and the lactation period, the ration should be supplemented with material rich in vitamin B_1, such as dried brewer's yeast, baker's yeast, wheat germ, roe, liver or malt extract. (*F. Ender and A. Helgebostad. Eksperimentell beriberi hos sölvrev. Skandinavisk Veterinär-Tidskrift, xxix, December 1939, pp. 1232-1244.*)

Potassium Iodide in Fowl Paralysis (=Lymphomatosis)

Duodenal or cecal coccidia and *Streptococcus viridans* isolated in the great nerve trunks were found invariably to be associated with fowl paralysis. Worm eggs, coccidial oöcysts and the original streptococci fed to experimental fowls resulted in paralysis in one of them, and the streptococcus was isolated from the great nerve trunks post mortem.

As the streptococcus was suspected of being the cause of the paralysis, an attempt was made to destroy it with drugs or by raising the leucocytic defenses. It was thought the leucocytes might be stimulated to remove the lymphocytes, found, post mortem, to be infiltrating the nerves and

organs of typical cases. The literature is sparse in references to systematic chemotherapy of the disease.

Three drugs were tried: The proprietary drug, antibacsyn, reputed to cause leucocytosis in bacterial infections; sulfanilamide; and potassium iodide. Only the latter proved to give beneficial results, and these were striking in several instances. It was given in doses of 5 cc. (1.25 dr.) of a 10 per cent solution intraperitoneally, on two successive days. Out of eight paralyzed fowls thus treated, five made uneventful recoveries without relapse. This experimental work was well controlled and tables showing the effects of the drugs on the leucocyte count are given. *(Ernest Gray. Some Experiments upon the Therapeutic Treatment of Fowl Paralysis and the Value of Iodine in Relieving the Symptoms of Such Cases. The Veterinary Journal, xcv, January 1940, pp. 28-34.)*

BOOK NOTICES

Anatomy of the Sheep's Brain

The author of *Anatomy of Animal Types*, E. A. Briggs of the University of Sydney, Australia, using the sheep's brain as a convenient type for the study of the mammalian encephalon, adds a valuable brochure on the gross anatomy of the brain not only of sheep but also of other domestic mammals.

The treatise is intended for students entering the course in zoölogy. It is anatomy in its purest form; there is no mention of function. The directions for dissecting a brain in order to expose its component structures are well told and will be found informative for the further study of the more minute details. Four excellent figures in the text and eight plates add materially to the value of the book. In short, the subject is covered as well as possible in a few pages. *(E. A. Briggs, D.Sc., assistant professor of zoölogy, University of Sidney. Angus & Robinson, Sydney, Australia, 1939. Price, 6 shillings.)*

The Kosher Code of the Orthodox Jew

Researches into the history of medicine have in recent years disclosed facts on the antiquity of systematic meat inspection that establish important data on the veterinary service of the oldest known civilizations, on a service governed by strict laws which were set aside through many centuries and revived only when the world began to awake from the sadistic hypnosis of the Dark Ages—when the chaos of abandoned science and philosophy and orderly government characteristic of the ancient nations broke down.

The Kosher Code gathers from the Babylonian Talmud, the Bible and other sources a wealth of details which call for a revision of our modernistic ideas on this and other hygienic practices. In distinguishing *kasher* from the nonedible portion of the body, one is reminded that anatomy is an old science, not a study born in the Italian school of the Renaissance. Moreover, the harmfulness of the organs compared with the flesh, as pointed out, shows the keen insight of ancient hygienists on the components of the body of animals that are apt to transmit disease to the consumer.

The Laws of Terefah, which deal with the conditions that disqualify animals and their meat as to their fitness for food, contain details one finds in modern regulations on meat inspection. The disposal of the jaws, pharynx, larynx, trachea, bronchi, lungs, heart, liver, spleen, omentum, intestine, stomach, uterus, fetus, kidneys, brain, spinal cord, gall bladder, feet, legs, skin, ribs and vertebrae and other bones, joints, and lesions and infestations of these, were all subject to these old laws which the authors have conveniently brought between the covers of a book for the edification of those interested in the origin of dietary regulations of our period.

Terefah (defects) is defined as "torn by beasts," evidently at first for describing pastoral animals attacked by lions or other predatory beasts and left mangled but alive. But, obviously, the laws written under this name as taught in the Babylonian academies for three centuries applied to any vital injury or disease. Thus, many kinds

of terefahs were described in making up the proscriptions we know best as the Mosaic injunctions, or Biblical command on edible and nonedible (*kasher*) meat.

The remarkable thing about this book is the wealth of little-known details on meat inspection which were written into the laws of ancient people. The student of veterinary history and the meat inspector interested in the origin and antiquity of his art will be fascinated by the contents of this tome. *(The Kosher Code. By S. I. Levin, senior rabbi of Minneapolis, and Edward A. Boyden, professor of anatomy, University of Minnesota. The University of Minnesota Press. 243 pages. Price, $4.50.)*

Animal Sex Control

The information presented in *Animal Sex Control* has as a premise the work of Prof. Felix Unterberger of Germany. In 1930, Unterberger offered the thought that the sex of the offspring can be controlled by the selective action of an acid or alkaline medium on the sperm cells which possess the sex-determining chromosomes. Alkalizing the vagina with a sodium bicarbonate solution produces a high percentage of males, and acidification with lactic acid, the reverse.

Unterberger's results with the female of *Homo sapiens* should interest the dictators. Work conducted in England tends to substantiate Unterberger's findings. Through the use of the aforementioned alkali, 73 boys resulted from 74 conceptions. The English workers report an accuracy of 84 per cent.

The bulk of the actual experimental work reported in the text was sponsored by the *New York Daily News*. Rats and rabbits were the experimental animals and impressive results were obtained. It is unfortunate, however, that there is no mention of confirmatory work done in other laboratories. Data on dogs are meager and the same may be said of the work reported on cattle and horses. The results of this method as applied to dogs and cattle were reported by breeders and not by the laboratory which conducted the rat and rabbit experiments for the *News*. The information pertaining to horses, which was submitted by a veterinarian, is rather meager.

Even though there is a paucity of well-grounded data in respect to cattle, horses, dogs and, apparently, the human, the idea is fascinating, and of possible far-reaching economic importance provided that Unterberger's work can be adequately substantiated.

The book is written for the layman, but if the scientifically educated desire to learn how the alkali-acid method is applied, the information is in the text. Illustrations are numerous and a glossary of terms and an index are included. *(Animal Sex Control. Carl Warren, B.A., B.S., M.S.J. Orange Judd Publishing Co., New York, 1940. 167 pages. Price, $1.75.)*

The Boxer

Comes another addition to the Judy series, *The Boxer*, which, obedient to the orthodoxy of dog-book writing, sets down material on the generalities of canine management that is as applicable to one breed as to another.

Books on breeds of dogs that "stick to the text" are as rare as the proverbial teeth of the barnyard hen. Perhaps, the outlet for a book confined to the lovers of any one breed would not justify the expense of publication. So, in addition to the classical treatment of the breed itself, in go the stereotyped chapters on canine history, general anatomy, reproduction, genetics, pathology, hygiene, showing, shipping, etc., etc., required to make up a sizeable volume. While none of this material is actually redundant, it does, more or less, submerge the title and always cajoles the unsuspecting reader into believing that the author is a master mind on all things canine, which connoisseurs of other breeds may question.

The breeder of Boxers and Mastiffs telling the expert on tiny toys what to do may miss some of the finer points in toy-dog production. Moreover, that eternal introduction of so many dog books telling the world about the remote origin of the dog

might be left to the domain of common knowledge. *The Boxer,* for example, says, "The origin of the dog is shrowded in mystery," that the dog of the realm descended from the jackal, wild dog or wolf, and that it was the first animal to be domesticated. Why not leave such things to the archeologist, who does not regard the origin of the dog as being any more

—Reproduced from "The Boxer."

A Bullenbeiszer of 350 years ago.

mysterious than other units of the animal kingdom? Certainly, a writer on the Holstein cow would not feel obligated to relate the pre-history of *Bos taurus.* The gap between the origin of the species and of the breed of any animal is a stretch of time too long and dark to torment the naturalist as much as it does the writer on dogs. [Pardon the sarcasm, for *The Boxer* is otherwise a masterpiece.]

The author traces the Boxer back 350 years. It is the offspring of the Bullenbeiszer of German literature, and is not as others have said, a cross between the Great Dane and the English Bulldog. The Boxer is older than the latter breed and its fame lies in possessing the make-up of the bulldog type without retaining the fighting mentality. [It was one of the war dogs of the World War.] The history of the Boxer in Germany and in this country, where its popularity has increased tremendously in recent years, is fascinating reading and the diagrams on profile showing official standards are unusually revealing for the breeder.

The author disapproves the trimming of ears and the docking of tails by laymen and relates an incident wherein a breeder after waiting for two years for a litter lost seven pups from hemorrhage following amputation of their tails. The sketches and description of correct ear trimming for Boxers is worth the price of the book to any veterinarian, independent of the fact that the book throughout advises dog owners to seek professional advice for surgical and medical attention.

Owners are admonished not to wait until dogs are moribund before taking them to the veterinarian, who nowadays is available everywhere. In this connection, "give the doctor a break" is the author's theme. Vaccination against distemper is recommended as 90 per cent preventive, and vitamin B_1 is the remedy given for fright disease, the nature of which is regarded as

—Reproduced from "The Boxer."

Int. Ch. Check v. Hunnenstein—first Boxer to win an American B-i-s, and whose value, from the point of view of bloodlines, is only now becoming apparent.

still unknown. No mention is made of the researches of Elvehjem and Arnold, and Patton. *(The Boxer. By Dan M. Gordon, M.D., Judy Publishing Company, Chicago. Illustrated. 160 pages. Price, $2.50.)*

Science News Letter, the weekly commentor of science, is available to all members of the Association at the reduced rate of $3.00. The regular price is $5.00 per year. Address inquiries to the A.V.M.A. office, Chicago. Do not send any money as Science Service, Inc., will bill you.

THE NEWS

A.V.M.A. Activities

The Association and the profession will be represented officially at the coming United States Pharmacopoeial Convention, to be held in Washington in May of this year. This is the first time that the veterinary profession has been recognized by this body. President Way has appointed H. E. Moskey, Washington, D. C.; D. W. Ashcraft, Columbus, Ohio; and R. L. Mundhenk, Auburn, Ala., as delegates.

The convention is composed of delegates representing the various professions, medical schools, pharmacy colleges, state medical associations, certain research institutions, industrial groups, and other closely allied interests. Its principal purpose is to revise the Pharmacopoeia of the United States. Revision is handled by committees during the ten-year interval between conventions. Interim revisions are made from time to time to meet changing conditions and to cope with advances in scientific knowledge.

• • •

Not the least honor conferred upon a writer on national welfare is to have his writings published in the *Congressional Record*, as was done with the article on the veterinary service by Executive Secretary Merillat which appeared in the February 1940 issue of the *American Legion Magazine*. Many will suspect that the honor thus conferred is the work of Hon. George W. Gillie, congressman from Indiana, who thereby placed on record for all time to come a documentation of the part veterinary medicine plays in national health and economics.

• • •

Arrangements are being made to show, during a general session of the Washington meeting, the sound film, "Know Your Money," which has been produced by the U. S. Secret Service as an exposé of currency counterfeiting and government check forgery.

• • •

Technicolor motion pictures of the veterinary exhibit at the 1939 New York World's Fair, prepared under the sponsorship of the Association and allied organizations in coöperation with the American Museum of Health, are now available for showing at a limited number of veterinary meetings. The film is of the convenient 16-mm. size and runs approximately five minutes. Among the scenes appearing prominently are those dealing with veterinary research, eradication of cattle ticks and tuberculosis, control of hog cholera, inspection of milk and meat and related industrial developments, progress in controlling encephalomyelitis, a modern small animal hospital, and food value of meat and its use in normal, gaining and reducing diets. The film also shows groups of visitors viewing the exhibit.

• • •

President Way attended the convention of the American Foundation of Veterinary Therapy at Kansas City, Mo., March 15-16. A complete report of the proceedings of the meeting will be published in the May issue.

• • •

Executive Secretary Merillat attended the Alabama conference at Auburn, Ala., March 20-23, and spoke on "Modern Horse Practice."

• • •

Assistant Executive Secretary Ingmand spoke over the air at Hot Springs, Ark., while attending the Arkansas state meeting, March 21-22, on the importance of competent veterinary service. During the Iowa state meeting at Des Moines, January 23-25, he gave a similar radio address on "Animal Health Today." Dr. Ingmand also attended the Northwestern Wisconsin meeting on March 27.

• • •

D. M. Campbell of the Committee on Public Relations and Executive Secretary Merillat addressed the Agricultural Club of Chicago recently on the veterinary service of the United States.

• • •

One of the most comprehensive projects ever inaugurated by the Association is the taking of a complete census of the veterinary profession. Since the beginning of March, hundreds of the questionnaire forms specially prepared for this purpose have been mailed every day from the executive office. Within a short time the results of this important survey will be available.

• • •

Election of a member of the Executive Board for district 7 (Alaska, Hawaii, Idaho, Montana, Nebraska, North Dakota, Oregon, Philippine Islands, South Dakota, Washington and Wyoming) is now in progress. The candidates are W. J. Butler, H. E. Kingman, A. K. Kuttler, Charles Seagraves and W. T. Spencer.

Chairman Jakeman of the Board of Governors wishes to remind the members of the House about the importance of studying the contents of the new administrative by-laws and constitution, and to send suggestions for changes to the office of the Association so that all of the recommendations may be printed and redistributed before the time of the Washington session.

APPLICATIONS

First Listing*

ABELL, GUY F.
Rome, Pa.
V.M.D., University of Pennsylvania. 1935.
Vouchers: D. LaGrange and S. A. Peck.

ANDERSON, FRANK ALBERT
155 W. Grand Ave., Chicago, Ill.
D.V.M., Iowa State College, 1932. Vouchers: W. A. Young and Eugene B. Ingmand.

ANDRESS, WALTER MILLARD
4th and King Sts., Lewes, Del.
V.M.D., University of Pennsylvania, 1937.
Vouchers: Roy F. Davenport and Harry McDaniel.

BAYARD, RICHARD S.
New Milford, Conn.
V.M.D., University of Pennsylvania, 1929.
Vouchers: G. E. Corwin and C. P. Hines.

BRIGGS, RALPH COREY
Webster St., No. Hanover, Mass.
V.M.D., University of Pennsylvania, 1931.
Vouchers: L. A. Paquin and F. L. Briggs.

CASHELL, IRVING GERING
2807 18th St., N.W., Washington, D. C.
V.M.D., University of Pennsylvania, 1938.
Vouchers: J. P. Turner and I. M. Cashell.

CAUGHMAN, JR., FREDERICK PORTER
900 Harden St., Columbia, S. Car.
D.V.M., Alabama Polytechnic Institute, 1937.
Vouchers: R. L. Mundhenk and L. A. Merillat.

COX, N. A.
Box 1101, Lubbock, Texas.
D.V.M., Texas A. & M. College, 1937. Vouchers: W. A. Lawrence and F. G. Harbaugh.

CULLISON, JAMES M.
Charleston Veterinary Hospital, Charleston, Mo.
D.V.M., Iowa State College, 1938. Vouchers: A. E. Bott and H. E. Curry.

DEAN, EVARD ARTHUR
3121 S. Sherman St., Englewood, Colo.
D.V.S., Colorado State College, 1911; M.D., Gate City Medical College, 1914; P.G., University of Lemberg, Austria, 1916. Vouchers: George H. Glover and I. E. Newson.

DENNEWITZ, HOMER ARRESTA
4615 41st St., N. W., Washington, D. C.
D.V.M., George Washington University, 1918.
Vouchers: William M. Mohler and John R. Mohler.

DILLON, HUGH C.
Marion Junction, Ala.
D.V.M., Alabama Polytechnic Institute, 1934.
Vouchers: R. L. Mundhenk and L. A. Merillat.

FASKEN, J. W.
533 Doreen St., Winnipeg, Manitoba.
B.V.Sc., Ontario Veterinary College, 1916.
Vouchers: R. H. Lay and Alfred Savage.

FISHER, SAMUEL S.
507 Federal Bldg., Little Rock, Ark.
D.V.M., Michigan State College, 1936. Vouchers: John H. Morton and Eugene B. Ingmand.

FOY, GORDON L.
932 E. Hyde Park Ave., St. Joseph, Mo.
D.V.M., University of Georgia, 1932. Vouchers: George H. Murphy and William H. Bailey.

GARRETT, LA RUA
Tuscaloosa, Ala.
D.V.M., Alabama Polytechnic Institute, 1937.
Vouchers: R. L. Mundhenk and L. A. Merillat.

HIGGINS, WILLIAM YOUNG
46 Park Ave., Flemington, N. J.
D.V.M., Cornell University, 1934. Vouchers: Elvin R. Coon and J. R. Porteus.

ISBELL, W. J.
Auburn, Ala.
D.V.M., Alabama Polytechnic Institute, 1930.
Vouchers: R. L. Mundhenk and F. P. Woolf.

JEROME, NORMAN
Box 631, Route 2, San Diego, Calif.
B.V.Sc., Ontario Veterinary College, 1936.
Vouchers: W. L. Curtis and A. P. Immenschuh.

JOHNSON, SANFORD E.
3030 Newport St., Omaha, Neb.
D.V.M., Kansas State College, 1935. Vouchers: F. O. Garrett and Eugene B. Ingmand.

JONES, WILLIAM EDWARD
409 P. O. Bldg., Montgomery, Ala.
D.V.M., Alabama Polytechnic Institute, 1938.
Vouchers: R. L. Mundhenk and Hubert L. Allen.

KEARLEY, RICHARD I.
Andalusia, Ala.
D.V.M., Alabama Polytechnic Institute, 1914.
Vouchers: R. L. Mundhenk and L. A. Merillat.

KENDRICK, LEON RYAN
205 Circle Ave., Charlotte, N. Car.
D.V.M., Alabama Polytechnic Institute, 1913.
Vouchers: Wm. Moore and A. A. Husman.

KOCH, MEYER
General Delivery, Tuscaloosa, Ala.
D.V.M., Ohio State University, 1938. Vouchers: R. L. Mundhenk and L. A. Merillat.

*See January 1940 JOURNAL.

MACASAET, VALENTIN
Canlubang, Laguna, P. I.
D.V.M., University of the Philippines, 1923. Vouchers: A. K. Gomez and Vicente Ferriols.

MEADOWS, MARION E.
Phenix City, Ala.
D.V.M., Alabama Polytechnic Institute, 1912. Vouchers: R. L. Mundhenk and T. A. Sigler.

MEREDITH, CLARENCE PAUL
705 Main, Joplin, Mo.
D.V.M., Iowa State College, 1935. Vouchers: Eugene B. Ingmand and H. E. Curry.

NASH, JOHN G. C.
211 N. A. St., Oskaloosa, Iowa.
D.V.M., Kansas City Veterinary College, 1917. Vouchers: A. H. Quin and Eugene B. Ingmand.

O'BRIEN, JAMES H.
96 Summer St., Taunton, Mass.
B.V.Sc., Ontario Veterinary College, 1912. Vouchers: L. A. Paquin and Dennis S. Shannon.

OUTWATER, GEORGE ELWOOD
183 W. Thames St., Norwich, Conn.
V.M.D., University of Pennsylvania, 1934. Vouchers: G. E. Corwin and R. L. Smith.

PHELPS, GUY JULIUS
108 No. Lawrence St., Montgomery, Ala.
D.V.M., Alabama Polytechnic Institute, 1911. Vouchers: R. L. Mundhenk and L. A. Merillat.

PINKERTON, HARRY EDGAR
1519 Third Ave. North, Fort Dodge, Ia.
D.V.M., Ohio State University, 1905. Vouchers: H. C. Smith and A. H. Quin.

PLATT, WILLIAM BARRY
Wharton Ave., Somerville, N. J.
D.V.M., Michigan State College, 1935. Vouchers: J. R. Porteus and R. A. Hendershott.

RANNEY, CLARENCE L.
2456 Heliotrope Drive, Santa Ana, Calif.
D.V.M., Cornell University, 1934. Vouchers: W. L. Curtis and G. W. Closson.

ROSSMAN, LEWIS C.
Pelham, Ga.
D.V.M., University of Georgia, 1933. Vouchers: R. L. Mundhenk and L. A. Merillat.

SCHMIDT, MARTIN GEORGE
Box 42, Falmouth, Ky.
D.V.M., Iowa State College, 1936. Vouchers: Chas. E. Hagyard and A. H. Davidson.

SENDER, LUDWIG
c/o Fort Dodge Serum Co., Fort Dodge, Iowa.
D.V.M., Iowa State College, 1937. Vouchers: H. C. Smith and A. H. Quin.

SHAW, W. SUMNER
Millbrook, N. Y.
D.V.M., Cornell University, 1931. Vouchers: C. P. Zepp and Cassius Way.

SUEBSAENG, CHARAS
2300 Kalorama Road, N. W., Washington, D. C.
D.V.M., University of the Philippines, 1931.
M.S., Cornell University, 1939. Vouchers: William M. Mohler and Ervin A. Eichhorn.

TANNER, HAL W.
Eutlaw, Ala.
D.V.M., Alabama Polytechnic Institute, 1938. Vouchers: R. L. Mundhenk and W. E. Cotton.

TOLLEY, ARCHIE D.
1501 S. Nevada Ave., Colorado Springs, Colo.
D.V.M., Colorado State College, 1934. Vouchers: E. N. Stout and C. C. Fobenstein.

WHITE, KENNETH A.
Deary, Idaho.
D.V.M., State College of Washington, 1930. Vouchers: E. M. Gildow and R. L. Griffith.

Second Listing

Allen, Jap C., 320 Agricultural Bldg., Raleigh, N. Car.
Baxter, Joe Ford, Box 104, Jacksonville, N. Car.
Beaver, Russel Spencer, Harlan, Iowa.
Bertels, Floyd William, Lakefield, Minn.
Blackner, Reuben, Lyman, Wyo.
Bradley, Frederick, 314 Court St., Plymouth, Mass.
Buehler, Cleo J., 225 N. Main, Morton, Ill.
Burg, Nathan Walter, c/o Dr. N. O. Olson, P. O. Box 412, Coeur d'Alene, Idaho.
Campbell, H. L., 706 South Nile St., Tuskola, Ill.
Conover, Fred H., 702½ N. Evans St., Bloomington, Ill.
Cooperrider, Donald Elmer, 2174 W. 103rd St., Cleveland, Ohio.
Daniels, John F., Perry, Ill.
Darst, Lloyd M., Princeton, Ill.
Dollahite, James Walton, Marfa, Texas.
Dotson, Harry Fleming, Room 26, Live Stock Exchange Bldg., Wichita, Kan.
Flanigan, Jean A., Eugene, Ore.
Frock, Irvin Warner, Manchester, Md.
Gilbert, Darius Weed, 11 Highland Ave., Cohasset, Mass.
Green, Jr., George William, Easton, Md.
Grossman, Roger, 5635 University Ave., Chicago, Ill.
Hanna, Charles Gaylord, Scranton, Iowa.
Helyar, James Edwin, 4 Rutgers St., Stelton, N. J.
Henderson, Joseph Ansel, Aukeny, Iowa.
Hixson, Ralph, Falls City, Neb.
Hoyt, Harold G., Dundee, Ill.
Huston, Sherbern S., 2723 Arlington Ave., Davenport, Iowa.
Jones, Thomas John, 138 Milledge Terrace, Athens, Ga.
Judson, William Warren, 513 San Bernardo Ave., Laredo, Texas.
Kahn, Karl, 403 Palmyra Apts., 5111 S. University Ave., Chicago, Ill.
Kirkpatrick, Joseph Irl, Sedgwick, Kan.
Koll, Carl Henry, 2030 27th St., Des Moines, Iowa.

Leber, George W., 7th and Union, Pacific, Mo.
Lee, Jesse D., Box 527, Salmon, Idaho.
Legner, Armand A., Sandwich, Ill.
Long, E. I., 110 N. Olive, Wellington, Kan.
Loomis, Frank G., Yorkville, Ill.
Marsh, Earl E., 533 S. Douglas, Springfield, Ill.
McGrath, William Bernard, U. S. Soldiers' Home, Washington, D. C.
Merrick, James R., 5732 West Cermak Rd., Cicero, Ill.
Moser, Herman L., Pontiac, Ill.
Norton, Elwin Sheldon, Box 973, Meridian, Miss.
Nye, Russell O., Buda, Ill.
Old, Claude T., Sikeston, Mo.
Osburn, Melvin William, Independence, Iowa.
Packwood, Olin S., c/o Dr. Fred C. Driver, Post Office Bldg., Bismarck, N. Dak.
Patton, Glen Frank, 2705 N. 13th St., Kansas City, Kan.
Pennell, Benjamin Nixon, 843 Montauk Ave., New London, Conn.
Pirie, James William, 1108 Glass Road, Cedar Rapids, Iowa.
Price, Edmund R., P. O. Box 52, Twin Falls, Idaho.
Proctor, Lester, Hazelton, Iowa.
Prophitt, James Henry, Suffield, Conn.
Raudabaugh, W. G., Piper City, Ill.
Riker, Jr., Joseph Thaddeus, Box 1213, Klamath Falls, Ore.
Robinson, Virgil Benton, Connaway Hall, University of Missouri, Columbia, Mo.
Rodabaugh, H. D., 216 North Marion St., Kirksville, Mo.
Ruder, Frederick G., 300 North Pleasant St., Amherst, Mass.
Ryle, C. G., 340 North Broadway, Georgetown, Ky.
Schulz, Carl William, 716 East High, Jefferson City, Mo.
Sherwood, William James, Box 522, Washington, N. Car.
Sivyer, Roland Eugene, 5935 N. Montana Ave., Portland, Ore.
Smith, John Ray, Fairbury, Ill.
Stigers, Arthur Leroy, West Des Moines, Iowa.
Suits, Franklin H., Odessa, Mo.
Sundberg, A. Laurence, Laurens, Iowa.
Swanson, A. C., Webster City, Iowa.
Thomsen, Lloyd Henry, Gladbrook, Iowa.
Todd, Jr., William C., 697 Pennington Ave., Trenton, N. J.
Van Ausdall, Charles A., Edinburg, Ill.
Walters, F. A., 405 Singer Ave., Lemont, Ill.
Watkins, Ernest St. John, 1229 Chestnut St., San Francisco, Calif.
Wessels, Herbert P., 520 N. First St., Geneva, Ill.
Wilcox, Lee A., Washburn, Ill.

The amount that should accompany an application filed this month is $8.75. This includes membership fee and dues to January 1, 1941, including subscription to the JOURNAL.

Educational Campaign on Dog-Food-Testing Program

The educational campaign on the A.V.M.A. and the A.A.H.A. dog-food-testing program is bringing highly satisfactory results. For the past several months speakers have explained the purposes of the program before veterinary meetings in all parts of the country. Enthusiastic comments on these talks have been received. The profession is eager to learn more and to coöperate with the program.

"These educational talks have been the spark that started a considerable discussion among members of the staff on the diet of dogs," one member wrote after hearing one of these addresses. "Any and all instructions and references you can give us on the subject of diet will be appreciated. We hope that we will have the pleasure of your presence at another meeting soon again. The January meeting was the most interesting and instructive I have ever attended."

In these addresses by prominent members of the profession it is emphasized that every manufacturer of dog foods may have his products tested, in the hope of obtaining the seal of approval, by simply applying to the committee on foods of the American Animal Hospital Association and fulfilling certain requirements. At the present time five dog foods have been tested and granted the seal.

Veterinarians throughout the country are being told that they can recommend with confidence that the dog foods bearing the seal of approval are nutritionally adequate. It would not be possible for a manufacturer to submit a nutritionally inadequate food and obtain the seal. An acceptable food is one that is nutritionally adequate for growth and maintenance.

The speakers have emphasized also that the members of the scientific advisory council—who supervise the tests—and the committee on foods of the A.A.H.A. serve in the public interest without compensation for professional or administrative services. When the seal has been granted to a food, efforts are made to ascertain whether the quality is maintained. This is accomplished through retests during each year.

On different occasions veterinarians have been urged to warn their clients that scraps from the table may be very poor food for their pets. It has been found that scraps may be manifestly deficient in one or more necessary elements and that, therefore, pet owners should not rely upon scraps as the only food for their animals.

It also has been pointed out that many commercial dog foods are compounded carefully by expert dietitians and chemists and are manufactured by reliable companies. On the other hand, there appears to be a large number of dog foods of very poor quality on the market, all sorts of waste products being employed in their manufacture. In some instances this is

done in order to sell the foods at a low price. One must remember that it is not often possible to select a nutritious food for a dog from the labels on containers or from advertising claims. Both may be so misrepresented and inaccurate that they are meaningless.

This program merits the support of veterinarians, of dog owners and of the legitimate, honest manufacturers of dog foods. It is in the interest of his client that the veterinarian should urge the dog owner to ask for foods bearing the seal of approval. By so doing, the dog owner is certain to give his dog the life-sustaining and growth-promoting food it needs.

New Advertisers

The attention of the readers is called to five new advertisers. For many years the Fromm Bros. Co. of Hamburg and Grafton, Wis., has been engaged in breeding and raising foxes and mink, and today are regarded as one of the largest producers in the industry. Early in the life of this firm it was found that disease was a serious deterrent to the successful operation of their ranches. Thus, it was necessary to build extensive laboratories and provide a competent staff to cope with the many disease problems threatening their business. Through the years the Fromm laboratory staff has contributed much to the knowledge of fox and mink diseases and of relatively recent date has developed effective biological products to control fox distemper and fox encephalitis. It was soon found that the fox-distemper-preventive agent could be adapted to the control of canine distemper. In the realization of the great importance of effectively controlling dog distemper, Fromm Laboratories, Inc., was formed to provide more extensive facilities for experimental work to make the product on a commercial scale.

Lehn & Fink, makers of the famous household disinfectant, Lysol, announce a new all-purpose disinfectant, Amphyl, for veterinary use. Free samples of this new product are available to members of the profession upon application to the manufacturer.

Balorations, Inc., a division of Tioga Mills, Waverly, N. Y., advertise their Ti-O-Ga puppy food. It is a dry food and enjoys a splendid reputation among dog owners and veterinarians alike. Special franchises are open to veterinarians.

VioBin Corporation of Chicago, Ill., producers of a quality cold-pressed extracted wheat germ oil, advertise for the first time in the current issue.

The National Drug Company, Philadelphia, took space for the first time in the March number, and a like amount of space is again devoted to National encephalomyelitis vaccine in this issue.

Veterinary Associations Laud Serum Producers' Program

The educational campaign being conducted *via* newspapers and radio among livestock owners by the Associated Serum Producers has won the plaudits of state and regional veterinary associations throughout the country. Realizing the tremendous benefit that is accruing to the veterinary profession through the producers' publicity program, several prominent associations have adopted resolutions expressing their appreciation of these efforts. At other conventions officers and individual members have presented verbal reports of the excellent results obtained.

U. S. GOVERNMENT

Progress in Bang's Disease Eradication

The federal bureau of animal industry recently announced the practical eradication of Bang's disease in 209 counties of 17 states. The regulations provide that whenever the percentage of dairy and breeding cattle over 6 months of age which have been tested does not exceed 1 per cent, nor the number of infected herds more than 5 per cent of the total number of herds in the area, such a county can be declared a modified accredited Bang's disease-free area, provided that all infected herds are kept in quarantine. Cattle in the infected herds must be retested at intervals of 30 to 90 days until all of them pass two consecutive negative tests and an additional negative test not less than six months after the second test. The reactors are removed from the herds and disposed of in accordance with the state and federal regulations. At present, the federal payment averages $14.50, the state payment, $16.20 and the salvage, $34.90.

Equine Encephalomyelitis, 1935-1939

Figures gathered by Shahan, Giltner and Schoening show that the incidence of encephalomyelitis in horses was as follows: 1935—23,000, 1936—4,000, 1937—170,000, and 1938—79,000.

For 1939, the number of cases was nominal. In fact, the disease seemed to have vanished. Extensive vaccination with formalized chick-embryo vaccine in districts where the disease had been prevalent is believed to have been a factor in lowering the morbidity, though unknown influences may have intervened.

During 1939, 9,462,305 doses of encephalomyelitis vaccine (chick) were produced and it is estimated that no less than 3,000,000 horses and mules received preventive treatment.

The mortality for the year 1935, 1936, and

1937 was about 25 per cent. In 1938, it dropped to 21 per cent.

In 1929, in 24 states where only western type virus was found or suspected, the mortality was 26.7 per cent. The mortality in nine states where only the eastern type virus was found or suspected was 89 per cent.

In addition to the horse, it is well to recall that the susceptible species are: **Western virus**—man, guinea pig, rat, mouse, rabbit, monkey, pigeon, calf, dog, goat, duck, hen, gratlag groose, hawk, European blackbird, tawny vulture, stork, mallard duck, ground squirrel, turkey, and guinea fowl; **Eastern virus**—cat, sheep, ring-necked pheasant, quail, pig and European hedgehog.

While the disease was rampant in the United States during 1935-1938, other countries were simultaneously affected, namely: Canada, South America, Russia, Japan and India. To an American scientist (Wyckoff) goes the credit of discovering an effective preventive.

Eichhorn on a Trip of Mercy

On February 24, Adolph Eichhorn left New York on the Manhattan for Genoa, Italy, and thence by rail to Budapest, Hungary. His services were loaned by the federal bureau of animal industry to the U. S. Public Health Service for the purpose of taking to Hungary a large quantity of two kinds of typhus-fever vaccine for use among the refugees in the Carpathian Mountains.

One of these vaccines was perfected by Dr. Cox of the U. S. Public Health Service and is prepared from chick-embryo virus along lines similar to the encephalomyelitis vaccine. The other type was prepared by Dr. Zinsser of Harvard University and is a tissue-culture vaccine for use as a prophylactic against typhus fever. The Lederle Laboratories, Inc., have prepared an equal quantity of the latter product for comparative tests in Hungary.

The National Institute of Health was desirous of obtaining the temporary services of Dr. Eichhorn for this trip because of his familiarity with the Hungarian language, the customs of the people, and the sections of the country involved. He also is a personal friend of Professor Johan, director of the Hungarian Hygienic Institute at Budapest, under whose immediate supervision the relative values of the two forms of typhus-fever vaccine will be tested on human beings and the results recorded.

J. R. M.

The American Bison

According to the U. S. bureau of biological survey, there are 5,029 bison in the United States and Alaska. Shortly after the turn of the century the American bison was in grave danger of extinction.

B. A. I. Transfers

Birtwhistle McCormack from Sioux City, Iowa, to Kansas City, Kan., on virus-serum control.

Harold Burstein from Wilmington, Del., to Philadelphia, Pa., on meat inspection.

Forrest O. Cox from Chicago, Ill., to South St. Joseph, Mo., on meat inspection.

Joseph S. Gordon from Sioux City, Iowa, to Omaha, Neb., on virus-serum control.

Glen C. Harrington from Omaha, Neb., to Denver, Colo., on meat inspection.

Louis F. Heydecker from South St. Paul, Minn., to Kansas City, Kan., on meat inspection.

Paul G. Palmer from Austin, Minn., to Portland, Ore., on meat inspection.

Richard E. Pflugrad from New York, N. Y., to East Berkshire, Vt., in charge of meat inspection.

John R. Pooley from Sioux City, Iowa, to Prairie du Chien, Wis., in charge of meat inspection.

Charles F. Prchal from Richmond, Va., to Des Moines, Iowa, on Bang's disease.

Irwin H. Roberts from Raleigh, N. Car., to Kansas City, Kan., zoölogical division.

Arthur L. Tellejohn from Omaha, Neb., to Columbus, Ohio, on virus-serum control.

Albert W. Vornheder from Atlanta, Ga., to Kansas City, Kan., on meat inspection.

Retirement.—Robert O. Rothermel, Philadelphia, Pa., January 31, 1940.

Deaths.—Paul H. Hartman, Philadelphia, Pa., February 10, 1940; Frederick H. Thompson, Portland, Ore., February 27, 1940.

Regular Army

Captain Don L. Deane is relieved from further assignment and duty as a student, Medical Field Service School, Carlisle Barracks, Pa., effective upon completion of the present course of instruction, on or about March 9, 1940, and will then return to his proper station, the Army Medical Center, Washington, D. C., and report to the commanding general for duty.

Captain Walter Smit is relieved from assignment and duty at the Army Medical Center, Washington, D. C., and from temporary duty at Carlisle Barracks, Pa., effective upon completion of the present course of instruction at the Medical Field Service School, on or about March 9, 1940, and is then assigned to duty at Carlisle Barracks, Pa., for duty.

Captain Stephen G. Asbill is relieved from assignment and duty at the Army Medical Center, Washington, D. C., and from temporary duty at Carlisle Barracks, Pa., effective upon completion of the present course of instruction at the Medical Field Service School, on or about March 9, 1940, and assigned to duty at Fort Bliss, Texas. Captain Asbill will proceed

to Fort Sheridan, Ill., for approximately four months of temporary duty prior to going to Fort Bliss.

Announcement is made of the promotion of 1st Lieut. Don L. Mace to the grade of captain, with rank from February 10, 1940.

Major Frank M. Lee, now on duty at Plattsburg Barracks, N. Y., is assigned to duty at Jefferson Barracks, Mo., effective March 10, 1940, and to additional duty in the office of the quartermaster, St. Louis, Mo.

Captain George T. Price is assigned to duty at Fort Bragg, N. Car., upon his return from foreign service in the Panama Canal department.

Tne following veterinary reserve officers (all first lieutenants) have been called to active duty in connection with the air corps expansion program and assigned to the stations opposite their names for duty: Charles E. Robinson, Sioux City, Iowa—Lowry Field, Colo.; Harold M. Deane, So. San Francisco, Calif.—Seattle Q.M.D., Wash.; Wm. E. Eggert, Jr., Wortendyke, N. J.—N. Y. Port of Emb., Brooklyn, N. Y.

First Lieut. William S. Gochenour, Jr., veterinary corps reserve, who has been on active duty at Camp Joseph T. Robinson, Ark., since December 1939, is assigned to duty at Selfridge Field, Mich. Lieut. Gochenour will be on temporary duty at Jefferson Barracks, Mo., until about March 12, 1940, before going to Selfridge Field.

In connection with the increase of the army by 17,000, directed by the President under his limited emergency proclamation, Public, No. 415, 76th Congress, authorized calling to active duty additional reserve officers other than those previously authorized by the air corps expansion act. To date the following veterinary reserve officers have been ordered to active duty and assigned to stations indicated after their names, for a period of one year: 1st Lieut. Burton C. Thomson, Fayetteville, N. Car.—Ft. Bragg, N. Car.; and 1st Lieut. Robert L. Alkire, Laurel, Md.—Fort Dix, N. J.

Lieut. Colonel Peter T. Carpenter, now at Fitzsimons General Hospital, Denver, Colo., is relieved from his present assignment and duty at Fort Logan, Colo., and directed to proceed to his home and await retirement.

Captain Wesley W. Bertz is relieved from assignment and duty at Carlisle Barracks, Pa., effective on or about March 11, 1940, and assigned to duty in the Philippine department. He will proceed to Washington, D. C., and report to the surgeon general for temporary duty in his office until time to proceed to New York, N. Y., and sail on or about June 8, 1940, for the Philippine department.

Captain Robert N. Earhart, veterinary corps reserve, who has been on active duty at Fort Des Moines, is relieved from that station effective on or about March 1, 1940, and directed to proceed to Kelly Field, Texas, for duty.

Veterinary Corps Reserve

NEW ACCEPTANCES—FIRST LIEUTENANTS

Lester Arvel Deese, 808 W. Suwannee St., Fitzgerald, Ga.

Charles Henry Failing, 60 W. Burdick St., Oxford, Mich.

Howard Carter Poulin, 12½ Prospect St., Nashua, N. H.

Harvey Louis Rubin, P. O. Box 481, Auburn, Ala.

Richard David Siegle, P. O. Box 94, Gen'l Delivery, Alto, Mich.

Dale Robert Stephenson, Marion, Mich.

Benners Brasfield Vail, Jr., Armstrong, Ala.

Alan William Winter, 205 E. Dewey Ave., Buchanan, Mich.

PROMOTIONS

To Captain.—Trevor Hugh Hughes, 38½ Delaware St., Cooperstown, N. Y.; William Moses Thompson, 1203 White, Grand Junction, Colo.

To Major.—George Donald Loder, 6606 N. Campbell Ave., Portland, Ore.

AMONG THE STATES

California

Effective February 23, all dogs and cats in San Francisco are under a rigid six-month quarantine by order of the state department of public health. The measure was adopted following a human death resulting from the scratch of a cat, although a total of 31 cases of animal rabies occurring in San Francisco since June 1939 was cited as the principal reason for the quarantine.

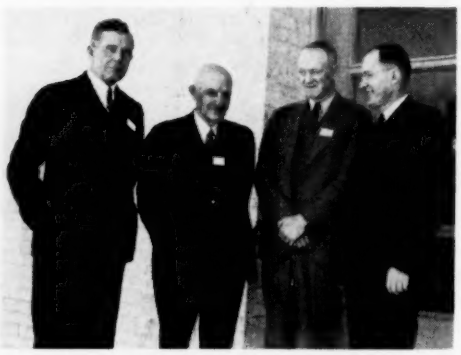
Under the state department's action, four rules will be enforced: 1) No cat or dog may be taken away from the premises of its owner unless it is on a leash. The leash must not be longer than 6 ft. and must be in the hands of a person over 15 years of age. 2) All dogs and cats are barred from street cars, buses and other public conveyances. 3) No dog or cat may be taken out of the city and county of San Francisco or brought into the city without written permission from the office of the director of public health. 4) Animals quarantined as rabies contacts must be confined to the owner's premises or other place designated by the health department for a period of three months, or they will be destroyed.

• • •

Colorado

In the January 1940 issue (p. 126) it was stated that four Colorado veterinarians each have a son studying veterinary medicine at Colorado State College. To the names listed should be added that of W. G. Blake (Colo. '11), who also has a son enrolled in the College.

The Fourth Annual Short Course for Veterinarians was convened at Colorado State College, Fort Collins, February 19-22. The evening of Tuesday, February 20, was given over to the dedication of the new veterinary building, with the following program comprising the ceremony: "Address of Welcome," Charles A. Lory, president of the College; "The Public Works Administration," David R. Kennicott, regional director of the WPA; "Larimer County Medical Society," E. Miner Morrill, secretary; "Colorado Veterinary Medical Association," Jay H. Bouton, secretary; "The Veterinary School," George H. Glover, dean emeritus of the Division of Veterinary Medicine, Colorado State College; "The Veterinarian in the Conquest of Human Disease," W. H. Feldman of The Mayo Foundation, Rochester, Minn.; "A. V. M. A. Progress—Past, Present and Future," Cassius Way, president of the national association.



Left to right: Cassius Way, president of the national association; George H. Glover, dean emeritus of the veterinary division, Colorado State College; William H. Feldman of The Mayo Foundation, Rochester, Minn.; and I. E. Newsom, dean of the veterinary division, Colorado State College.

Dean I. E. Newsom presided at the ceremony.

District of Columbia

The District of Columbia Veterinary Medical Association held its first quarterly meeting of the year at the Mayflower Hotel in Washington, D. C., on the evening of February 14, H. W. Schoening presiding.

Officers were elected for the ensuing year as follows: Col. R. A. Kelser, president; Harry A. Locke, first vice-president; A. E. Wight, second vice-president; and Wm. M. Mohler, secretary-treasurer (reëlected).

Cassius Way, president of the national association, who was the guest of the evening, spoke on "A. V. M. A. Matters." John R. Mohler, chairman of the local Committee on Arrangements for the coming A. V. M. A. meeting, gave a detailed progress report of the Committee's activities. A lengthy discussion followed his report, and a motion was passed to contribute $50 to the Committee.

Although a severe blizzard gripped the District of Columbia throughout the day, 50 members were in attendance to greet Dr. Way. Refreshments were served to conclude the program.

WM. M. MOHLER, *Secretary.*

Georgia

The Atlanta Veterinary Medical Society held its regular monthly meeting on March 12. Bang's disease control was discussed at length by W. C. Dendinger and J. M. Sutton. It was pointed out that Georgia ranks second among all the states of the Union in Bang's disease accreditation work.

CHAS. C. RIFE, *Resident Sec'y.*

Illinois

The 58th annual meeting of the Illinois State Veterinary Medical Association was held in Springfield, February 15-16, with nearly 500 veterinarians and their wives in attendance.

The literary sessions were planned to appeal principally to practitioners, with the following comprising this portion of the two-day program:

The Duties of an Accredited Veterinarian—H. C. Rinehart, chief veterinarian, Springfield.

The Rabies Problem—E. B. Ingmand, assistant executive secretary of the A.V.M.A., Chicago.

Illegal Veterinary Practice in Illinois—H. B. Sill, investigator in the state department of registration and education, Springfield.

Artificial Insemination of Dairy Cattle—R. L. Anderes, Kansas City, Mo.

So-Called Shipping Fever of Dairy Cattle—W. C. Glenney, Elgin.

Regular Monthly Examination of Herds to Control Disease and Increase Profits—C. H. Case, Akron, Ohio.

Diseases of Feeder Cattle—W. S. O'Neal, St. Charles, Mo.

Nutritional Diseases of Live Stock (illustrated by technicolor sound film, entitled "Vitamins on Parade," produced by the University of Wisconsin)—Cliff D. Carpenter, Fort Wayne, Ind.

The Veterinarian and the Control of Poultry Diseases—F. C. Tucker, Claypool, Ind.

What the Practitioner Can Best Do for the A.V.M.A.—Cassius Way, president of the national association, New York, N. Y.

The Rôle of the Veterinarian in Effective Immunization Against Hog Cholera with Tissue Vaccine—W. H. Boynton, University of California, Berkeley, Calif.

A Small Animal Practice in the Country—Byron P. Merrick, Berlin Heights, Ohio.

Practical Points in Canine Practice—R. Merrick, Cicero.

The dinner session was devoted entirely to entertainment, dancing and renewing acquaintances. President W. B. Holmes of Springfield acted as master of ceremonies.

A feature of the business session was the report of the special legislative committee appointed to rewrite the veterinary practice act of Illinois. The report stimulated a lively discussion and brought out many interesting views.

Officers elected for the ensuing year are J. W.

Lucas of Abingdon, president; D. E. Sisk of Mansfield, vice-president; and C. C. Hastings of Williamsville, secretary-treasurer.

Iowa

Much interest is centered in the coöperative state-federal Bang's disease blood-testing program to be launched on or about May 1. All private practitioners of the state will participate in the project at a scale of compensation in keeping with professional standards.

A. H. QUIN, *Resident Sec'y.*

Louisiana

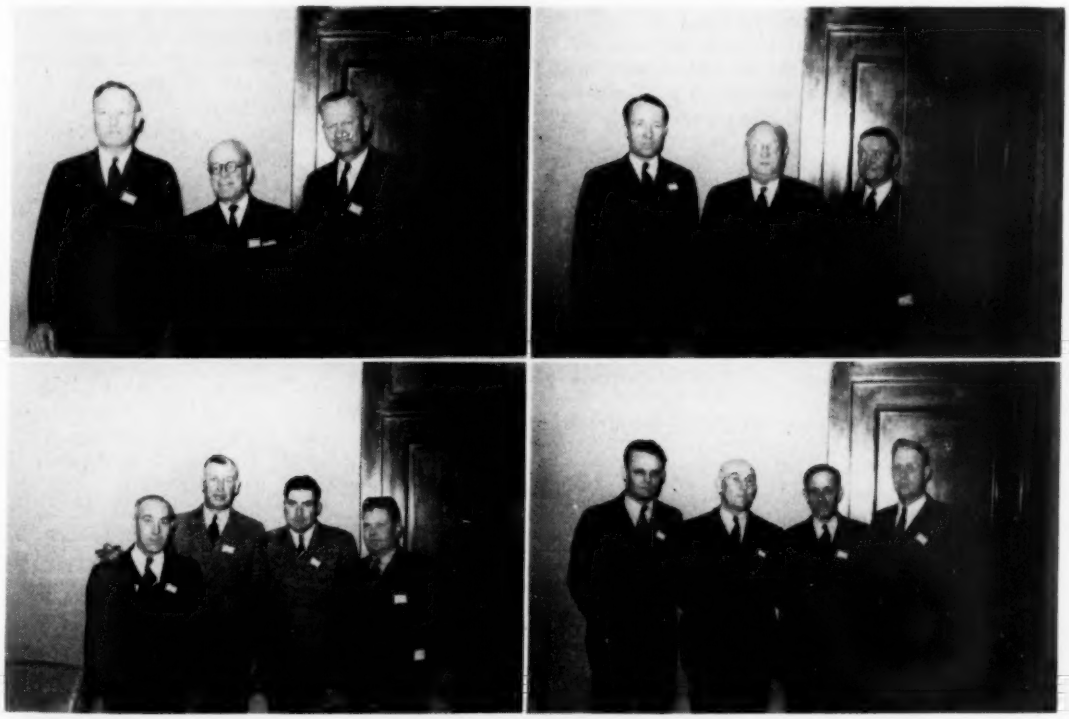
The Louisiana Veterinary Medical Association held its annual meeting in conjunction with the ninth annual short course at the Dalrymple building, Louisiana State University, Baton Rouge, February 7-8, W. A. McDonald of Baton Rouge presiding.

James Farquharson of Colorado State College, Fort Collins, Colo., spoke on "Sulfanilamide in Veterinary Practice" and "New Technics in Veterinary Surgery," the latter being demonstrated with lantern slides and motion pictures. S. W. Haigler of St. Louis, Mo., presented papers on puppy problems and skin diseases of dogs.

The afternoon of the second day was devoted to demonstrations, with Dr. Haigler in charge of the small animal section and Dr. Farquharson supervising the large animal section.

A. V. Young of Shreveport gave a paper on "Heartworms of Dogs"; W. C. Schofield of the veterinary department of Louisiana State University presented "The Importance of Poultry Diseases to the Veterinary Practitioner"; Harry Gayden of the department of animal industry at the state university spoke on "Minerals in Livestock Feeding"; J. C. Miller of the same department discussed the value of live stock in the South; and T. Y. Gladney, M.D., spoke on undulant fever, referring frequently to the work of Dr. Huddleson of Michigan State College and other veterinarians with reference to the diagnosis of this disease.

Photos from the JOURNAL'S Candid Camera



ILLINOIS VETERINARIANS

Upper left (left to right): C. C. Hastings of Williamsville, secretary of the Illinois state association; W. B. Holmes of Springfield, immediate past-president of the Illinois organization; and J. W. Lucas of Abingdon, newly elected president. Upper right: C. N. Bramer, Evanston; L. A. Merillat, Chicago; and C. L. Lehman, Flanagan. Lower left: H. R. Hornbaker, Galesburg; Ross Carter, Alexis; L. M. Darst, Princeton; and W. C. Glenney, Elgin. Lower right: H. P. Wessels, Geneva; A. G. Gieske, Barrington; W. W. Warnock, Aledo; and C. A. Cox, Fithian. (Photographs taken at the 58th annual meeting of the Illinois State Veterinary Medical Association, Springfield, Ill., February 15-16, 1940.)

J. D. Jones of Bastrop was elected president for the ensuing year; F. F. McNeely of Colfax, vice-president; and C. M. Heflin of Baton Rouge, secretary-treasurer (reëlected). J. Arthur Goodwin of New Iberia was elected delegate to the House of Representatives of the national association and H. H. Baur of Monroe, alternate.

Col. Troy H. Middleton, vice-president and comptroller of the state university, gave an excellent address at the banquet on the evening of February 7. M. H. Gandy acted as toastmaster. During the course of the banquet, the state association presented E. P. Flower of Baton Rouge, state veterinarian since 1908, with an enlarged portrait of himself.

C. M. HEFLIN, *Secretary.*

Michigan

The regular winter short course for veterinarians was held at Michigan State College, East Lansing, January 23-25, with about 150 in attendance. Among the guest speakers were A. H. Schmidt of Triumph, Minn.; R. G. Green of the medical school, University of Minnesota, St. Paul, Minn.; C. C. Hastings of Williamsville, Ill.; Geo. E. Bowler, Ann Arbor; H. W. Emerson of the Pasteur Institute, Ann Arbor; O. M. Gruhzit of Detroit; E. J. McLachlan of the department of health, Jackson; and H. D. Peet of the Peet Packing Company, Chesaning.

C. F. CLARK, *Resident Sec'y.*

Nevada

At the annual meeting of the Nevada State Veterinary Association, held at Reno, February 23, G. T. Woodward of Fallon was elected president; R. A. Given of Reno, vice-president; and Warren B. Earl of Reno, secretary-treasurer (reëlected). Edward Records and Lyman R. Vawter, both of the University of Nevada at Reno, were elected delegate and alternate, respectively, to the House of Representatives of the national association.

WARREN B. EARL, *Secretary.*

New Hampshire

The New Hampshire Veterinary Medical Association held their annual meeting on February 28 and elected the following officers for the ensuing year: H. M. Lewis of Nashua, president; H. T. Paul of Portsmouth, vice-president; and C. L. Martin of Durham, secretary-treasurer.

The meeting consisted mainly of a discussion led by R. W. Smith, state veterinarian, of Bang's disease testing and the New Hampshire plan of control and eradication of Bang's disease.

C. L. MARTIN, *Secretary.*

North Carolina

In conjunction with the North Carolina State College at Raleigh, the North Carolina State Veterinary Medical Association held its second annual veterinary conference on January 22-25, with approximately 60 per cent of the veterinarians of the state attending, in spite of a severe snowstorm. Largely responsible for the excellent program and arrangements were A. A. Husman of Raleigh, president of the Association, and C. D. Grinnells of the state college.

On the opening day of the meeting, officials of the college welcomed the veterinarians. J. B. Hollenbeck of Columbus, Ohio, discussed various diseases of swine and made special reference to swine erysipelas. Following, a swine clinic was held.

During the remaining three days, motion pictures on bovine and equine pregnancy were presented by Charles E. Fanslau of New York City; W. L. Boyd of the University of Minnesota, St. Paul, Minn., discussed various cattle problems and diseases and conducted a clinic; Carl F. Schlotthauer of the The Mayo Foundation, Rochester, Minn., presented papers on "Nervous Diseases of Lower Animals" and "Tumors of the Mammary Gland of the Bitch," and also conducted a small animal clinic; W. J. Dann of Duke University at Durham gave an excellent paper on blacktongue, covering the history and recent developments in treating this malady; L. D. Frederick of Chicago, Ill., read a well-grounded paper on canine nutrition; and, through the courtesy of The Ohio State University, a motion picture of the bovine rumen as seen through a rumenal fistula was shown.

Mark Welsh of College Park, Md., presided over the banquet held in the dining hall of the state college, during which State Commissioner of Agriculture Kerr Scott and his assistant, Mr. Coltrane, each made a short talk.

J. H. BROWN, *Secretary.*

North Dakota

A conference on Bang's disease and tuberculosis eradication was held on February 6-7 at West Fargo and Fargo. The program was arranged by Fred C. Driver of Bismarck, inspector-in-charge for North Dakota. The session was attended by federal bureau of animal industry and practicing veterinarians of the state.

The meeting served to coördinate the activities of veterinarians engaged in Bang's disease work and bovine and avian tuberculosis eradication. Reports presented at the conference showed that 27,140 herds totaling 372,329 head of cattle have been tested for Bang's disease in the state since 1934. About one per cent of the cattle tested in area work now in progress in 26 counties were reported as positive reactors. There are 2,153 flocks of poultry under federal supervision in North Dakota, it was stated, and approximately 50 per cent of the flocks and 10 per cent of all the birds are infected with avian tuberculosis.

Ohio

The 13th annual conference of the College of Veterinary Medicine, The Ohio State University, Columbus, will be held June 12-14. This year, as formerly, the program is quite extensive and includes some of the foremost veterinarians and educators in America. The entire three-day period will be devoted to the discussion of topics of vital importance to every veterinarian, and particularly to the practitioner.

South Carolina

South Carolina is beginning to have an unusually early spring, after about three or four weeks of the most severe winter the state has had in over half a century. Considerable damage was done to the small grain crop but live stock apparently have not been affected by the low temperatures.

A number of breeders have moved into the state since last fall and have purchased large acreages for development into cattle growing. It would therefore appear that the veterinarians of South Carolina will have a good year.

W. A. BARNETTE, *Resident Sec'y.*

Virginia

The Virginia State Veterinary Medical Association held its winter meeting at the Hotel John Marshall in Richmond, February 8-9. This session was in accordance with an action taken at the 1939 annual meeting—"To take the place of the conference for graduate veterinarians sponsored heretofore by the veterinary staff of the Virginia Polytechnic Institute at Blacksburg, which is to give them a breathing spell." Fifty-one veterinarians were in attendance.

Among the host of speakers participating in the program were Adolph Eichhorn of the federal animal disease station, Beltsville, Md.; A. Henry Craige, Jr., of the University of Pennsylvania, Philadelphia, Pa.; and Frank Breed of Lincoln, Neb. One of the most interesting features of the session was a question box on swine erysipelas, conducted by Frank Breed.

A banquet was held, with more than 100 in attendance, including a number of state legislators who were guests of the members.

A. J. SIPOS, *Resident Sec'y.*

Wisconsin

At a meeting of the state Holstein breeders' association at Waukesha, February 16, Herbert Lothe discussed "Calfhood Vaccination from the Viewpoint of the Local Veterinarian" and W. Wisnicky of the College of Agriculture, University of Wisconsin, Madison, spoke on "Brucella Vaccination from the Viewpoint of Research."

JAMES S. HEALY, *Resident Sec'y.*

STUDENT CHAPTER ACTIVITIES

The New Constitution

The revised student chapter constitution is meeting with general approval. Although copies of the document were mailed less than a month ago, more than half of the chapters already have approved it and within the next 30 days the majority of them will be operating under the improved set-up. The acceptance of this model constitution will aid materially in unifying chapter activities.

One of the features of the new constitution is that it provides for a president-elect, which will enable the incumbent president to thoroughly acquaint his successor with the duties of the office. It also provides for two elections each collegiate year, thus permitting a larger number of members to hold offices. The provision for an executive board composed of the chapter officers and representatives from each class should add much to the stability of the organizations.

Diplomas

The new diplomas have apparently won favor, since orders from two chapters have been received already. Others have written that the document has been approved and that orders for the seniors will follow shortly. Inasmuch as the eligibility rules governing the issuance of diplomas include attendance at meetings and payment of dues in addition to other requirements, it is important that undergraduates, particularly freshmen and sophomores, take their student organization seriously, as exceptions will not be made later.

Student Keys

Junior students who are members in good standing of their respective chapters and are certain of being admitted to the senior classes next fall are now eligible to wear the student key. All orders must clear through the chapter president or person designated by him to handle requisitions for the tokens.

Colorado State College

An average of 120 members have attended each meeting of the Colorado organization.

A particularly interesting session was held on March 11, when Richard F. Bourne entertained the members with a lecture and demonstration on hypnotism. A number of students aided in the presentation, volunteering themselves to Dr. Bourne's hypnotic powers. The value of hypnotism as a therapeutic agent and some of the results obtained by its use were explained by Dr. Bourne.

"ROSY" ANDRUS, *Reporter.*

Texas A. & M. College

At the March meeting of the Texas chapter, films were shown on reproductive disorders in mares and cows and on artificial insemination.

Tentative arrangements have been made to show the film, "Ruminant Motility," at the April meeting and it is planned to have Ray Kay Daily, prominent oculist, appear on this program.

A chapter dance will be held on May 17 in Sbisa hall, and invitations are being extended to graduates of the College's veterinary school, members of the faculty, and members of the extension service. Various committees have been appointed by President Rodgers. It is expected that this event will attract veterinarians from all sectors, since it will be somewhat of a "homecoming" for the veterinary alumni of the College.

CHARLES V. ISAAC, *Secretary.*

Iowa State College

At a meeting of the Iowa chapter on February 28, President Emerson reported that the executive committee had adopted the new constitution prepared by the Association. A motion was made and carried that honorary members be elected to the chapter.

Ignatius Campagna, one of the members, performed a few slight-of-hand tricks, and Guest Speaker G. E. McFarland talked on "The Diseases of Animals That May Be Transmitted to Man."

WALTER TIMMS, *Secretary.*

Ontario Veterinary College

A feature of the Ontario chapter meetings during 1939 was the presentation of short papers written by student members. The contributors to these programs were as follows: E. W. Kreuger, E. L. Symington, J. S. Gaw, J. H. Leslie, E. Kading, N. Schmidt, S. Morrison, D. W. Thompson, C. Davey, W. B. Giesbrecht, N. H. McAninch, E. W. Manning, D. McDole, N. E. Anderson, L. R. Davidson, W. P. O'Connor, J. M. Warren, E. J. Meyer, N. Schollard, R. W. Ford, J. M. McKague, G. Cueva, H. Skelding, E. F. Pallister, M. R. Campbell, W. R. Morgan, G. L. Bannister, G. Schultz, J. P. Gilman, A. Taylor, G. Schiedel, D. C. Master, S. Thibaudeau, K. Burns, F. Codlin, G. I. Steele, R. Radmore, C. K. Hetherington, F. Clement, E. Knapp, D. C. Docksteader, W. G. Perrson, D. Garrick, D. Young, and J. E. Hendry.

Virtually every subject of interest in both large and small animal medicine was covered during the course of these programs.

On October 14, 1939, C. D. McGilvray gave an interesting talk on the relationship between the A.V.M.A. and the students. At present, the chapter has a membership of 60, including the officers.

JAMES J. FANNING, *Secretary.*

COMING MEETINGS

Small Animal Hospital Association. Los Angeles, Calif. April 2, 1940. R. W. Gerry, secretary, 8474 Melrose Ave., Los Angeles, Calif.

New York City, Veterinary Medical Association of. Hotel New Yorker, New York, N. Y. April 3, 1940. J. J. Merenda, secretary, 136 W. 53rd St., New York, N. Y.

Dallas-Fort Worth Veterinary Medical Society. Dallas, Texas. April 4, 1940. H. V. Cardona, secretary, 2736 Purington Ave., Fort Worth, Texas.

Houston Veterinary Association. Houston, Texas. April 5, 1940. W. T. Hufnall, secretary, 1612-14 E. Alabama Ave., Houston, Texas.

St. Louis District Veterinary Medical Association. Melbourne Hotel, St. Louis, Mo. April 5, 1940. J. P. Torrey, secretary, 610 Veronica Ave., East St. Louis, Ill.

Chicago Veterinary Medical Association. Hotel Sherman, Chicago, Ill. April 9, 1940. G. S. Elwood, secretary, 5449 Broadway, Chicago, Ill.

Southeastern Michigan Veterinary Medical Association. Medical Arts Bldg., 3919 John R St., Detroit, Mich. April 10, 1940. F. D. Egan, secretary, 17422 Woodward Ave., Detroit, Mich.

Kansas City Veterinary Medical Association. Kansas City, Mo. April 15, 1940. S. J. Schilling, secretary, Box 167, Kansas City, Mo.

San Diego County Veterinary Medical Association. Zoölogical Research Bldg., Balboa Park, San Diego, Calif. April 15, 1940. Paul D. DeLay, secretary, State Poultry Pathological Laboratory, Balboa Park, San Diego, Calif.

Southern California Veterinary Medical Association. Chamber of Commerce Bldg., Los Angeles, Calif. April 17, 1940. Charles Eastman, secretary, 725 S. Vancouver Ave., Los Angeles, Calif.

South Alabama-West Florida Veterinary Medical Association. Bonifay, Fla. April 19, 1940. E. M. Nighbert, secretary, Route 1, Box 118, Cantonment, Fla.

Southwestern Minnesota Veterinary Medical Association. City Hall, Lambert, Minn. April 22, 1940. L. E. Stanton, secretary, Jackson, Minn.

District of Columbia Veterinary Medical Association. Mayflower Hotel, Washington, D. C. April 23, 1940. Wm. M. Mohler, secretary, 5508 Nebraska Ave., N. W., Washington, D. C.

Keystone Veterinary Medical Association. School of Veterinary Medicine, University of Pennsylvania, Philadelphia, Pa. April 24, 1940. A. Henry Craige, secretary, University of Pennsylvania, Philadelphia, Pa.

Maine Veterinary Medical Association. University of Maine, Orono, Me. April 24, 1940. J. F. Witter, secretary, University of Maine, Orono, Me.

Massachusetts Veterinary Association. Hotel Westminster, Copley Square, Boston, Mass. April 24, 1940. H. W. Jakeman, secretary, 44 Bromfield St., Boston, Mass.

American Scientific Congress. Washington, D. C. May 10-16, 1940. (A. V. M. A. special committee to represent veterinary profession of United States: Col. R. A. Kelser, John R. Mohler and Willard H. Wright.)

Connecticut Veterinary Medical Association. Sage Park Race Track, Windsor Conn. May 16, 1940. Geo. E. Corwin, secretary, State Office Bldg., Hartford, Conn.

American Veterinary Medical Association. Mayflower Hotel, Washington, D. C. August 26-30, 1940. L. A. Merillat, secretary, 221 N. La Salle St., Chicago, Ill.

PERSONAL NOTES

Births

To Dr. (Iowa '37) and Mrs. E. T. Anderson of Kewanee, Ill., a daughter, Bonnie Lynn, February 12, 1940.

To Dr. (O.S.U. '31) and Mrs. Fritz Volkmar of Chicago, Ill., a son, Carl Fredrich, January 3, 1940.

To Dr. (K.S.C. '33) and Mrs. G. D. Allen of Little Rock, Ark., a son, Kenneth Wayne, March 8, 1940.

To Dr. (Wash. '31) and Mrs. E. M. Joneschild of Helena, Mont., a son, Edward David, January 1, 1940.

To Dr. (Iowa '36) and Mrs. W. C. Glenney of Elgin, Ill., a daughter, Judyth Lynn, February 21, 1940.

Marriages

W. H. Smeltzer, Jr. (Iowa '39) of Mendota, Ill., to Mary Knoebel of Chicago, Ill., January 19, 1940.

V. W. Ruth (Ont. '38) of Lansdale, Pa., to Mary Elizabeth Wallace of Akron, Ohio, December 9, 1939.

Activities

J. W. Robertson (Chi. '12) of Portsmouth, Ohio, was recently appointed to the Portsmouth board of health.

Herman Ladson (U.S.C.V.S. '10) of Rockville, Md., is recovering from a recent illness at the Washington Sanitorium in Takoma Park, Md.

E. E. Tobin (Colo. '13) of Greeley, Colo., president of the Colorado state association, has been confined to his bed with a serious illness for the past few weeks.

C. L. Everson (O.S.U. '29) has recovered from an attack of undulant fever and returned to the Maryland livestock sanitary service following an absence of five months.

M. W. Williams (A.P.I. '15), formerly of Center, Ala., has entered Alabama Polytechnic Institute at Auburn as a graduate student of veterinary medicine and part-time instructor in veterinary anatomy.

Arthur F. Schalk (O.S.U. '08) of The Ohio State University at Columbus was elected president of the Columbus board of health on February 13, 1940, after having served on the board for six years.

L. J. Tompkins (Corn. '12) of Packanack, N. J., veterinarian for the Sheffield Farms Co., was elected president of the Metropolitan Certified Milk Producers at their annual meeting in New York City, January 29, 1940.

E. M. Gildow (Wash. '22), who has been connected with the University of Idaho at Moscow for the past ten years as veterinarian in the department of animal husbandry, has accepted a position with the Carnation stock farms at Carnation, Wash.

E. G. Cole (Colo. '36) has resigned his office with the Salt Lake City, Utah, health department and is returning to Nebraska, where he formerly resided, to establish a practice. It is understood that Salt Lake City will abandon the services of a veterinarian.

William Martin Simpson (McG. '90) of Malden, Mass., prominent in the veterinary profession for over 50 years, celebrated his 76th birthday on February 9. Despite three major operations in as many years, Dr. Simpson is in apparently good health and keeps office and hospital hours daily.

W. J. Butler (N.Y.-Amer. '03), state veterinarian of Montana and secretary of the state livestock sanitary board, was honored in the January 1940 number of the *Montana Wool Growers News Letter*, which carried his likeness in full-page display. The picture is a copy of the portrait which hangs in the famous Saddle and Sirloin Club in Chicago.

H. Van Roekel (Iowa '25) of the Massachusetts State College at Amherst, is chairman of the pathology section of the coming 32nd annual meeting of the Poultry Science Association, to be held in Ithaca, N. Y., June 26-29, 1940. F. R. Beaudette (K.S.C. '19) of New Brunswick, N. J., is vice-chairman of the section and E. P. Johnson (Mich. '25) of Blacksburg, Va., is a member of the award committee.

Frank H. Barr (Chi. '92) of Albuquerque, N. Mex., was recently honored by the 111th cavalry of the New Mexico National Guard. In

recognition of his willing service the winter camp of the regiment, held in Albuquerque, was named after him and, on January 27, 1940, Dr. Barr, who was retired from active duty in December 1931 and placed upon inactive duty as a major, was presented with a colonelship on the governor's staff. At an informal reception after this ceremony, Colonel Barr was presented with his eagles by Captains T. I. Means (Colo. '20) and N. G. Cobbett (Colo. '21), the present veterinarians of the 111th cavalry.

DEATHS

Valentine M. Knapp of Danbury, Conn., died of a heart attack on February 22, 1940.

Dr. Knapp was a graduate of Cornell University, class of '04, and a member of the Association since 1906.

Paul H. Hartman of Philadelphia, Pa., died on February 10, 1940.

Born in Boyertown, Pa., on April 18, 1898, Dr. Hartman attended the University of Pennsylvania, from which he was graduated in 1922. He joined the Association in 1932.

Charles E. Stewart of Chariton, Iowa, died following a protracted illness on January 15, 1940, at the age of 79.

Born at Hebron, Ohio, March 12, 1860, Dr. Stewart attended the Chicago Veterinary College, from which he was graduated in 1894. Along with Downing, Johnson, Quin, Niles and Drinkwater, all now deceased, he helped to formulate and promote the passing of Iowa's first practice act. He joined the national association in 1902.

Robert Maxwell Walsh, 44, widely known veterinarian in the Southeast, died on February 5, 1940, at his home in Atlanta, Ga., after an illness of nine years.

Born in Cairo, Ga., June 22, 1895, Dr. Walsh was graduated from the Kansas City Veterinary College in 1916. For ten years prior to retiring in 1931, forced by ill health, he held the position of assistant state veterinarian of Georgia. He joined the national association in 1926, remaining an active member until his retirement, and was a regular attendant at all veterinary meetings in his state and section. He also was a Mason and a Shriner.

C. C. R.

Earl M. Keef, 58, of Sacramento, Calif., died on December 25, 1939.

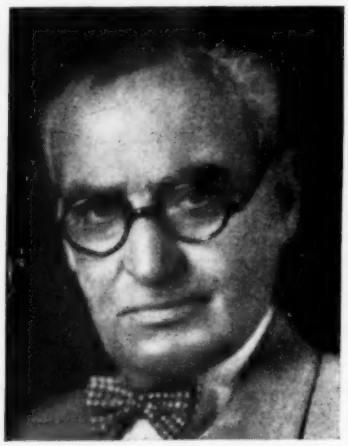
Born at San Lucas, Monterey county, Calif., November 20, 1881, Dr. Keef attended the San Francisco Veterinary College, from which he was graduated in 1913. For four years following graduation, he was engaged in private practice. In 1917, he entered the employ of the California state department of agriculture and, with the exception of one year, 1929, during which he took a leave of absence to assume the position of superintendent of a large dairy ranch in southern California, he served in the department until illness forced his retirement in 1936.

Dr. Keef joined the A.V.M.A. in 1915.

C. U. D.

John Francis De Vine, 65, of Goshen, N. Y., internationally known veterinarian and president of the J. F. De Vine Laboratories, Inc., died aboard the M. S. Kungsholm, near the island of Tahiti in the South Pacific, on March 3, 1940.

Born at Rhinebeck, N. Y., June 27, 1874, he attended the American Veterinary College



J. F. De Vine

from which he was graduated in 1898, after winning gold medals as writer of the best practical examination and the best theoretical examination. He was the only graduate ever to gain both medals.

Dr. De Vine had devoted himself principally to consultation and research since 1923. He was United States delegate to the Eleventh International Veterinary Congress at London in 1930, and at that time made a tour of Europe to lecture in leading veterinary schools.

As a writer, Dr. De Vine made innumerable contributions to the literature, perhaps the best known of which is his book, published in 1913, on "Bovine Tuberculosis in Relation to Human Tuberculosis."

From 1911 until 1921, he was professor of cattle pathology and veterinary obstetrics at New York University. In 1911, he served as president of the United States Live Stock Sanitary Association, and was president of the New York State Veterinary Medical Association in 1912.

Dr. De Vine was an active member of the national association since 1899 and will be remembered for his many distinctive contributions to its progress.

Equine Encephalomyelitis

The report of the Chief of the U. S. Bureau of Animal Industry dated January 20, 1940, shows that while the incidence of this disease was much less in 1939 than in the previous year, it was still sufficiently prevalent to constitute a real menace to the horse population if vaccination is neglected.

It is proper for veterinarians to bring to the attention of horse owners that the disease has now occurred in all of the states west of the Mississippi River, and that cases have been reported in one-third of all the counties of the United States. The report above referred to shows that more than 8,000 cases were reported to the Bureau in 1939.

Information now available indicates that the duration of immunity following the use of chick embryo type vaccine is usually eight months or more, but that after ten months many vaccinated animals are not immune. This emphasizes the desirability of annual vaccination in the spring.

The price of encephalomyelitis vaccine has recently been reduced 40%.

We shall be glad to furnish, free of charge, cards suitable for distribution to horse owners. These emphasize the desirability of early immunization and should help practitioners to get the bulk of this work out of the way before the rush incident to the immunization of spring pigs.

ASHE LOCKHART, Inc.

"Producers of Better Biologicals for Graduate Veterinarians."

800 Woodswether Road **Kansas City, Missouri**

In Canada—Canada Representatives, Ltd.
193-195 Spadina Avenue, Toronto, Ontario

MEMBER VETERINARY EXHIBITORS' ASSOCIATION

ENCEPHALOMYELITIS SPECIAL

Equine Encephalomyelitis Vaccine
(chick embryo origin)

The new, modern, fully equipped laboratory building devoted exclusively to the production of Equine Encephalomyelitis Vaccine is illustrated below. This up-to-date double unit structure located on the Jen-Sal Biological Farm, includes incubators—refrigerators—virus inoculating, harvesting, preparation, and sterilizing rooms for the production of both Eastern and Western vaccine. Test rooms and offices are located on the lower level.

High protective immunity against Equine Encephalomyelitis by vaccination with chick embryo vaccine has been demonstrated by classical laboratory experiments and by field experience.

". . . probaby no less than 3,000 000 horses and mules, (about one-fifth of the entire number) received specific prophylactic treatment (1939). Just how greatly this extensive application of vaccine retarded the spread of the disease is problematical, but it probably was one of the major factors."—*J. R. Mohler, Chief of the Bureau of Animal Industry, Washington, D. C.*

JENSEN-SALSBERY LABORATORIES, INC.
KANSAS CITY, MO.

PRINTING PRODUCTS CORPORATION—CHICAGO